```
=> e boman hans g/au
E1            2     BOMAN HANNA/AU
E2           32     BOMAN HANS/AU
E3          160 --> BOMAN HANS G/AU
E4            1     BOMAN HELENA/AU
E5           38     BOMAN HELGE/AU
E6            6     BOMAN I/AU
E7           32     BOMAN I A/AU
E8            2     BOMAN I ANITA/AU
E9            4     BOMAN I L/AU
E10         325     BOMAN J/AU
E11           1     BOMAN J A/AU
E12           3     BOMAN J E/AU

=> s e2-e3 and (l1-37)
L1            5 ("BOMAN HANS"/AU OR "BOMAN HANS G"/AU) AND (L1-37)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2            3 DUP REM L1 (2 DUPLICATES REMOVED)

=> d 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y


L2   ANSWER 1 OF 3  USPATFULL on STN
AN     2007:68445  USPATFULL
TI     Method for determining the susceptibility of a subject to infection
IN     Boman, Hans G., Stockholm, SWEDEN
       Andersson, Mats, Stockholm, SWEDEN
       Putsep, Katrin, Stockholm, SWEDEN
       Carlsson, Goran, Stockholm, SWEDEN
PI     US 2007059691       A1  20070315
AI     US 2003-530606      A1  20031010 (10)
       WO 2003-EP11240         20031010
                               20060221  PCT 371 date
PRAI   GB 2002-23655       20021010
DT     Utility
FS     APPLICATION
LN.CNT 1179
INCL   INCLM: 435/006.000
       INCLS: 435/007.320; 424/050.000
NCL    NCLM:  435/006.000
       NCLS:  435/007.320; 424/050.000
IC     IPCI   C12Q0001-68 [I,A]; G01N0033-554 [I,A]; G01N0033-569 [I,A];
              A61K0008-96 [I,A]

L2   ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2004:333962  CAPLUS
DN   140:353243
TI   Method of diagnosis
IN   Boman, Hans; Andersson, Mats; Puetsep, Katrin; Carlsson, Goeran
PA   Mabtech Ab, Swed.
SO   PCT Int. Appl., 40 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2004034061        A2    20040422    WO 2003-EP11240         20031010
     WO 2004034061        A3    20040521
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE,
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            GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
            LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ,
            OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM,
            TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
            KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
            FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
            BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
    CA 2501926          A1    20040422    CA 2003-2501926        20031010
    AU 2003282027       A1    20040504    AU 2003-282027         20031010
    EP 1549957          A2    20050706    EP 2003-773640         20031010
        R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
           IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
    JP 2006502395       T     20060119    JP 2004-542487         20031010
    US 2007059691       A1    20070315    US 2006-530606         20060221
PRAI GB 2002-23655      A     20021010
    WO 2003-EP11240     W     20031010

L2   ANSWER 3 OF 3  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
     DUPLICATE 1
AN   2002:591930  BIOSIS
DN   PREV200200591930
TI   Deficiency of antibacterial peptides in patients with morbus Kostmann: An
     observation study.
AU   Putsep, Katrin; Carlsson, Goran; Boman, Hans G.; Andersson, Mats
     [Reprint author]
CS   Microbiology and Tumorbiology Center, Karolinska Institutet, S-171 77,
     Stockholm, Sweden
     mats.andersson@mtc.ki.se
SO   Lancet (North American Edition), (October 12, 2002) Vol. 360, No. 9340,
     pp. 1144-1149. print.
     ISSN: 0099-5355.
DT   Article
LA   English
ED   Entered STN: 13 Nov 2002
     Last Updated on STN: 13 Nov 2002


=> e andersson mats/au
E1            1     ANDERSSON MARTIN PJ/AU
E2            1     ANDERSSON MATHIAS/AU
E3          225 --> ANDERSSON MATS/AU
E4            5     ANDERSSON MATS B/AU
E5            1     ANDERSSON MATS C/AU
E6            1     ANDERSSON MATS GUNNAR/AU
E7            1     ANDERSSON MATS H/AU
E8            1     ANDERSSON MATS J/AU
E9            9     ANDERSSON MATS O/AU
E10          75     ANDERSSON MATS R/AU
E11           1     ANDERSSON MATS ROLAND/AU
E12           2     ANDERSSON MATS T/AU

=> s e3-e12 and (LL-37)
L3            5 ("ANDERSSON MATS"/AU OR "ANDERSSON MATS B"/AU OR "ANDERSSON
                MATS C"/AU OR "ANDERSSON MATS GUNNAR"/AU OR "ANDERSSON MATS
               H"/AU OR "ANDERSSON MATS J"/AU OR "ANDERSSON MATS O"/AU OR "ANDER
               SSON MATS R"/AU OR "ANDERSSON MATS ROLAND"/AU OR "ANDERSSON MATS
               T"/AU) AND (LL-37)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4            3 DUP REM L3 (2 DUPLICATES REMOVED)

=> d bib ab 1-
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YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L4 ANSWER 1 OF 3 USPATFULL on STN
AN 2007:68445 USPATFULL
TI Method for determining the susceptibility of a subject to infection
IN Boman, Hans G., Stockholm, SWEDEN
Andersson, Mats, Stockholm, SWEDEN
Putsep, Katrin, Stockholm, SWEDEN
Carlsson, Goran, Stockholm, SWEDEN
PI US 2007059691 A1 20070315
AI US 2003-530606 A1 20031010 (10)
WO 2003-EP11240 20031010
20060221 PCT 371 date
PRAI GB 2002-23655 20021010
DT Utility
FS APPLICATION
LREP HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US
CLMN Number of Claims: 32
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 1179
AB A method for determining the susceptibility of a subject to infection, which method comprises: (i) providing a sample from said subject; (ii) detecting any LL-37 present in said sample; (iii) optionally comparing the level of LL-37 in said sample to a control sample; and (iv) determining the susceptibility of said subject to infection, wherein no LL-37 or a low level of LL-37 indicates that said subject is susceptible to infection.

L4 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:333962 CAPLUS
DN 140:353243
TI Method of diagnosis
IN Boman, Hans; Andersson, Mats; Puetsep, Katrin; Carlsson, Goeran
PA Mabtech Ab, Swed.
SO PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004034061	A2	20040422	WO 2003-EP11240	20031010
	WO 2004034061	A3	20040521		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2501926	A1	20040422	CA 2003-2501926	20031010
	AU 2003282027	A1	20040504	AU 2003-282027	20031010
	EP 1549957	A2	20050706	EP 2003-773640	20031010
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	JP 2006502395	T	20060119	JP 2004-542487	20031010
	US 2007059691	A1	20070315	US 2006-530606	20060221
PRAI	GB 2002-23655	A	20021010		

WO 2003-EP11240 W 20031010

AB A method is disclosed for determining the susceptibility of a subject to infection, which method comprises: (i) providing a sample from said subject; (ii) detecting any LL-37 present in said sample; (iii) optionally comparing the level of LL-37 in said sample to a control sample; and (iv) determining the susceptibility of said subject to infection, wherein no LL-37 or a low level of LL-37 indicates that said subject is susceptible to infection.

L4 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
AN 2002:591930 BIOSIS
DN PREV200200591930
TI Deficiency of antibacterial peptides in patients with morbus Kostmann: An observation study.
AU Putsep, Katrin; Carlsson, Goran; Boman, Hans G.; Andersson, Mats [Reprint author]
CS Microbiology and Tumorbiology Center, Karolinska Institutet, S-171 77, Stockholm, Sweden
mats.andersson@mtc.ki.se
SO Lancet (North American Edition), (October 12, 2002) Vol. 360, No. 9340, pp. 1144-1149. print.
ISSN: 0099-5355.
DT Article
LA English
ED Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002
AB Background Antibacterial peptides, such as defersins and LL-37, are natural bactericidal components similar in potency to classic antibiotics. These peptides are produced at mucosal linings in the body and the skin, and by leucocytes such as neutrophils and natural killer cells. Patients with morbus Kostmann-a severe congenital neutropenia-are treated by recombinant granulocyte-colony stimulating factor, which restores their levels of neutrophils. Despite this treatment, patients still have recurrent infections and periodontal disease. Our aim was to investigate if defensins and LL-37 are deficient in patients with morbus Kostmann. Methods We studied samples of neutrophils, plasma, and saliva from six patients with congenital neutropenia and 22 healthy controls for presence of antibacterial peptides. Neutrophils were analysed by high-performance liquid chromatography and mass spectrometry for alpha-defensins. All samples were analysed by western blot for cathelin-LL-37 (precursor of LL-37) and LL-37.
Neutrophils were also tested for lactoferrin and ability to produce oxidative burst. Findings Neutrophils from patients with morbus Kostmann were deficient in cathelin-LL-37 and had reduced concentrations of alpha-defensins HNP1-3. No cathelin-LL-37 could be detected in plasma and saliva from patients. One patient with morbus Kostmann who had had bone-marrow transplantation had almost normal concentrations of LL-37. Lactoferrin concentrations and oxidative burst were normal in all patients. All patients with morbus Kostmann had severe periodontal disease, apart from the individual who had had a bone-marrow transplant, whose dental status was normal. Interpretation Antibacterial peptides are a vital part of the first line of antibacterial immune defence. Deficiency in saliva LL-37 accords with occurrence of periodontal disease in patients with morbus Kostmann.

```
=> e putsep katrin/au
E1            5     PUTSEP E P/AU
E2           17     PUTSEP K/AU
E3           14 --> PUTSEP KATRIN/AU
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E4            1      PUTSEP V/AU
E5            6      PUTSEPP A/AU
E6            2      PUTSEV A I/AU
E7            2      PUTSEV I I/AU
E8           24      PUTSEVA N M/AU
E9            1      PUTSEY JAMES G/AU
E10           1      PUTSEYKO E K/AU
E11           2      PUTSEYS R/AU
E12          11      PUTSEYS ROLAND/AU

=> s e2-e3 and (LL-37)
L5            6 ("PUTSEP K"/AU OR "PUTSEP KATRIN"/AU) AND (LL-37)

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6             2 DUP REM L5 (4 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L6   ANSWER 1 OF 2  USPATFULL on STN
AN     2007:68445  USPATFULL
TI     Method for determining the susceptibility of a subject to infection
IN     Boman, Hans G., Stockholm, SWEDEN
       Andersson, Mats, Stockholm, SWEDEN
         Putsep, Katrin, Stockholm, SWEDEN
       Carlsson, Goran, Stockholm, SWEDEN
PI     US 2007059691       A1  20070315
AI     US 2003-530606      A1  20031010 (10)
       WO 2003-EP11240         20031010
                               20060221  PCT 371 date
PRAI   GB 2002-23655       20021010
DT     Utility
FS     APPLICATION
LREP   HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX
       9133, CONCORD, MA, 01742-9133, US
CLMN   Number of Claims: 32
ECL    Exemplary Claim: 1
DRWN   3 Drawing Page(s)
LN.CNT 1179
AB     A method for determining the susceptibility of a subject to infection,
       which method comprises: (i) providing a sample from said subject; (ii)
       detecting any LL-37 present in said sample; (iii)
       optionally comparing the level of LL-37 in said
       sample to a control sample; and (iv) determining the susceptibility of
       said subject to infection, wherein no LL-37 or a low
       level of LL-37 indicates that said subject is
       susceptible to infection.

L6   ANSWER 2 OF 2  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
     DUPLICATE 1
AN   2002:591930  BIOSIS
DN   PREV200200591930
TI   Deficiency of antibacterial peptides in patients with morbus Kostmann: An
     observation study.
AU   Putsep, Katrin; Carlsson, Goran; Boman, Hans G.; Andersson, Mats
     [Reprint author]
CS   Microbiology and Tumorbiology Center, Karolinska Institutet, S-171 77,
     Stockholm, Sweden
     mats.andersson@mtc.ki.se
SO   Lancet (North American Edition), (October 12, 2002) Vol. 360, No. 9340,
     pp. 1144-1149. print.
     ISSN: 0099-5355.
DT   Article
```

```
LA   English
ED   Entered STN: 13 Nov 2002
     Last Updated on STN: 13 Nov 2002
AB   Background Antibacterial peptides, such as defersins and LL-
     37, are natural bactericidal components similar in potency to
     classic antibiotics.  These peptides are produced at mucosal linings in
     the body and the skin, and by leucocytes such as neutrophils and natural
     killer cells.  Patients with morbus Kostmann-a severe congenital
     neutropenia-are treated by recombinant granulocyte-colony stimulating
     factor, which restores their levels of neutrophils.  Despite this
     treatment, patients still have recurrent infections and periodontal
     disease.  Our aim was to investigate if defensins and LL-
     37 are deficient in patients with morbus Kostmann.  Methods We
     studied samples of neutrophils, plasma, and saliva from six patients with
     congenital neutropenia and 22 healthy controls for presence of
     antibacterial peptides.  Neutrophils were analysed by high-performance
     liquid chromatography and mass spectrometry for alpha-defensins.  All
     samples were analysed by western blot for cathelin-LL-37
     (precursor of LL-37) and LL-37.
     Neutrophils were also tested for lactoferrin and ability to produce
     oxidative burst.  Findings Neutrophils from patients with morbus Kostmann
     were deficient in cathelin-LL-37 and had reduced
     concentrations of alpha-defensins HNP1-3.  No cathelin-LL-
     37 could be detected in plasma and saliva from patients.  One
     patient with morbus Kostmann who had had bone-marrow transplantation had
     almost normal concentrations of LL-37.  Lactoferrin
     concentrations and oxidative burst were normal in all patients.  All
     patients with morbus Kostmann had severe periodontal disease, apart from
     the individual who had had a bone-marrow transplant, whose dental status
     was normal.  Interpretation Antibacterial peptides are a vital part of the
     first line of antibacterial immune defence.  Deficiency in saliva
     LL-37 accords with occurrence of periodontal disease in
     patients with morbus Kostmann.


=> e carlsson goran/au
E1            8     CARLSSON GOETHE M/AU
E2            1     CARLSSON GOETHE MATS/AU
E3           82 --> CARLSSON GORAN/AU
E4            3     CARLSSON GOSTA/AU
E5           12     CARLSSON GOSTA E/AU
E6            1     CARLSSON GOTE A/AU
E7            1     CARLSSON GOTE ALLAN/AU
E8            1     CARLSSON GOTTFR/AU
E9            1     CARLSSON GRAFFMAN A C S/AU
E10           1     CARLSSON GRAN/AU
E11          22     CARLSSON GRANER U/AU
E12           9     CARLSSON GRANER ULLA/AU

=> s e3 and (LL-37)
L7            6 "CARLSSON GORAN"/AU AND (LL-37)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8            3 DUP REM L7 (3 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L8   ANSWER 1 OF 3  USPATFULL on STN
AN     2007:68445  USPATFULL
TI     Method for determining the susceptibility of a subject to infection
IN     Boman, Hans G., Stockholm, SWEDEN
       Andersson, Mats, Stockholm, SWEDEN
```

Putsep, Katrin, Stockholm, SWEDEN
Carlsson, Goran, Stockholm, SWEDEN

PI US 2007059691 A1 20070315
AI US 2003-530606 A1 20031010 (10)
WO 2003-EP11240 20031010
20060221 PCT 371 date
PRAI GB 2002-23655 20021010
DT Utility
FS APPLICATION
LREP HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US
CLMN Number of Claims: 32
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 1179
AB A method for determining the susceptibility of a subject to infection, which method comprises: (i) providing a sample from said subject; (ii) detecting any LL-37 present in said sample; (iii) optionally comparing the level of LL-37 in said sample to a control sample; and (iv) determining the susceptibility of said subject to infection, wherein no LL-37 or a low level of LL-37 indicates that said subject is susceptible to infection.

L8 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 1
AN 2007:126130 BIOSIS
DN PREV200700133550
TI Periodontal disease in patients from the original Kostmann family with severe congenital neutropenia.
AU Carlsson, Goran [Reprint Author]; Wahlin, Ylva-Britt; Johansson, Anders; Olsson, Anders; Eriksson, Torbjorn; Claesson, Rolf; Hanstrom, Lennart; Henter, Jan-Inge
CS Karolinska Univ Hosp, Dept Womens and Childrens Hlth, Childhood Canc Res Unit, S-17176 Stockholm, Sweden
goran.carlsson@ki.se
SO Journal of Periodontology, (APR 2006) Vol. 77, No. 4, pp. 744-751.
CODEN: JOPRAJ. ISSN: 0022-3492.
DT Article
LA English
ED Entered STN: 22 Feb 2007
Last Updated on STN: 22 Feb 2007
AB Background: Patients with Kostmann syndrome (severe congenital neutropenia [SCN]) typically normalize their absolute neutrophil count (ANC) upon granulocyte colony-stimulating factor (G-CSF) therapy. However, although they no longer experience life-threatening bacterial infections, they frequently still have recurrent gingivitis and even severe periodontitis, often starting in early childhood.Methods: We studied the periodontal disease in the four surviving patients belonging to the family originally described by Kostmann. Their odontological records, x-rays, color photos, bacterial cultures, serum antibodies to oral bacteria, and histopathological examinations were reviewed. The data were also correlated to previous investigations on their antibacterial peptides and molecular biology.Results: Three patients had periodontal disease, despite normal ANC and professional dental care, and had neutrophils deficient in antibacterial peptides. One of these patients also had a heterozygous mutation in the neutrophil elastase gene, had severe periodontal disease and overgrowth of the periodontal pathogen Actinobacillus actinomycetemcomitans in the dental flora, and 15 permanent teeth had been extracted by the age of 27. One bone marrow-transplanted patient had no periodontal disease.Conclusions: Normalized ANC levels are not sufficient to maintain normal oral health in SCN patients, and because neutrophils are important for first-line defense and innate immunity, the deficiency of the antibacterial peptide LL-37 probably explains

their chronic periodontal disease. Professional dental care is still important for SCN patients, despite treatment with G-CSF and normal ANC levels. Whether antibacterial peptides play a role in the pathogenesis of periodontitis in other patients remains to be elucidated.

L8 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
AN 2002:591930 BIOSIS
DN PREV200200591930
TI Deficiency of antibacterial peptides in patients with morbus Kostmann: An observation study.
AU Putsep, Katrin; Carlsson, Goran; Boman, Hans G.; Andersson, Mats [Reprint author]
CS Microbiology and Tumorbiology Center, Karolinska Institutet, S-171 77, Stockholm, Sweden
mats.andersson@mtc.ki.se
SO Lancet (North American Edition), (October 12, 2002) Vol. 360, No. 9340, pp. 1144-1149. print.
ISSN: 0099-5355.
DT Article
LA English
ED Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002
AB Background Antibacterial peptides, such as defersins and LL-37, are natural bactericidal components similar in potency to classic antibiotics. These peptides are produced at mucosal linings in the body and the skin, and by leucocytes such as neutrophils and natural killer cells. Patients with morbus Kostmann-a severe congenital neutropenia-are treated by recombinant granulocyte-colony stimulating factor, which restores their levels of neutrophils. Despite this treatment, patients still have recurrent infections and periodontal disease. Our aim was to investigate if defensins and LL-37 are deficient in patients with morbus Kostmann. Methods We studied samples of neutrophils, plasma, and saliva from six patients with congenital neutropenia and 22 healthy controls for presence of antibacterial peptides. Neutrophils were analysed by high-performance liquid chromatography and mass spectrometry for alpha-defensins. All samples were analysed by western blot for cathelin-LL-37 (precursor of LL-37) and LL-37.
Neutrophils were also tested for lactoferrin and ability to produce oxidative burst. Findings Neutrophils from patients with morbus Kostmann were deficient in cathelin-LL-37 and had reduced concentrations of alpha-defensins HNP1-3. No cathelin-LL-37 could be detected in plasma and saliva from patients. One patient with morbus Kostmann who had had bone-marrow transplantation had almost normal concentrations of LL-37. Lactoferrin concentrations and oxidative burst were normal in all patients. All patients with morbus Kostmann had severe periodontal disease, apart from the individual who had had a bone-marrow transplant, whose dental status was normal. Interpretation Antibacterial peptides are a vital part of the first line of antibacterial immune defence. Deficiency in saliva LL-37 accords with occurrence of periodontal disease in patients with morbus Kostmann.

```
=> s (LL-37)
L9        1571 (LL-37)

=> dup rem l9
PROCESSING IS APPROXIMATELY  94% COMPLETE FOR L9
PROCESSING COMPLETED FOR L9
L10         630 DUP REM L9 (941 DUPLICATES REMOVED)

=> s l10 and infection?
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L11 318 L10 AND INFECTION?

=> s l11 and susceptib?
L12 84 L11 AND SUSCEPTIB?

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 84 ANSWERS - CONTINUE? Y/(N):y

L12 ANSWER 1 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2007:145293 BIOSIS
DN PREV200700145257
TI Susceptibility of furunculosis-derived staphylococcus aureus to LL-37 and antibiotics.
AU Baranska-Rybak, W. [Reprint Author]; Sonesson, A.; Nowicki, R.; Schmidtchen, A.
CS Univ Gdansk, Dept Dermatol Venerol and Allergol, PL-80952 Gdansk, Poland
SO Journal of Investigative Dermatology, (AUG 2006) Vol. 126, No. Suppl. 3, pp. 96.
Meeting Info.: 36th Annual Meeting of the European-Society-of-Dermatology-Research (ESDR). Paris, FRANCE. September 06 -07, 2006. European Soc Dermatol Res.
CODEN: JIDEAE. ISSN: 0022-202X.
DT Conference; (Meeting)
Conference; (Meeting Poster)
LA English
ED Entered STN: 28 Feb 2007
Last Updated on STN: 28 Feb 2007

L12 ANSWER 2 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:670641 BIOSIS
DN PREV200600665908
TI Immunomodulatory properties of defensins and cathelicidins.
AU Bowdish, D. M. E.; Davidson, D. J.; Hancock, R. E. W. [Reprint Author]
CS Univ British Columbia, Ctr Microbial Dis and Immun Res, 232 Lower Mall Res Stn, Vancouver, BC V6T 1Z4, Canada
bob@cmdr.ubc.ca
SO Shafer, WM [Editor]. Curr. Top. Microbiol. Immunol., (2006) pp. 27-66.
Current Topics in Microbiology and Immunology.
Publisher: SPRINGER-VERLAG BERLIN, HEIDELBERGER PLATZ 3, D-14197 BERLIN, GERMANY. Series: CURRENT TOPICS IN MICROBIOLOGY AND IMMUNOLOGY.
CODEN: CTMIA3. ISSN: 0070-217X. ISBN: 3-540-29915-7(H).
DT Book; (Book Chapter)
LA English
ED Entered STN: 6 Dec 2006
Last Updated on STN: 6 Dec 2006
AB Host defence peptides are a conserved component of the innate immune response in all complex life forms. In humans, the major classes of host defence peptides include the alpha- and beta-defensins and the cathelicidin, hCAP-18/LL-37. These peptides are expressed in the granules of neutrophils and by a wide variety of tissue types. They have many roles in the immune response including both indirect and direct antimicrobial activity, the ability to act as chemokines as well as induce chemokine production leading to recruitment of leukocytes to the site of infection, the promotion of wound healing and an ability to modulate adaptive immunity. It appears that many of these properties are mediated though direct interaction of peptides with the cells of the innate immune response including monocytes, dendritic cells, T cells and epithelial cells. The importance of these peptides in immune responses has been demonstrated since animals defective in the expression of certain host defence peptides show greater susceptibility to bacterial infections. In the very few instances in which human patients have been demonstrated to have defective host defence peptide expression, these individuals suffer from frequent infections. Although studies of the immunomodulatory properties

of these peptides are in their infancy, there is a growing body of evidence suggesting that the immunomodulatory properties of these small, naturally occurring molecules might be harnessed for development as novel therapeutic agents.

L12 ANSWER 3 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:488251 BIOSIS
DN PREV200600491664
TI The antimicrobial peptide cathelicidin protects the urinary tract against invasive bacterial infection.
AU Chromek, Milan; Slamova, Zuzana; Bergman, Peter; Kovacs, Laszlo; Podracka, L'udmila; Ehren, Ingrid; Hokfelt, Tomas; Gudmundsson, Gudmundur H.; Gallo, Richard L.; Agerberth, Birgitta; Brauner, Annelie [Reprint Author]
CS Karolinska Univ Hosp, Dept Clin Microbiol, Microbiol and Tumorbiol Ctr, SE-17176 Stockholm, Sweden
Annelie.Brauner@ki.se
SO Nature Medicine, (JUN 2006) Vol. 12, No. 6, pp. 636-641.
ISSN: 1078-8956.
DT Article
LA English
ED Entered STN: 27 Sep 2006
Last Updated on STN: 27 Sep 2006
AB The urinary tract functions in close proximity to the outside environment, yet must remain free of microbial colonization to avoid disease. The mechanisms for establishing an antimicrobial barrier in this area are not completely understood. Here, we describe the production and function of the cathelicidin antimicrobial peptides LL-37, its precursor hCAP-18 and its ortholog CRAMP in epithelial cells of human and mouse urinary tract, respectively. Bacterial contact with epithelial cells resulted in rapid production and secretion of the respective peptides, and in humans LL-37/hCAP-18 was released into urine. Epithelium-derived cathelicidin substantially contributed to the protection of the urinary tract against infection, as shown using CRAMP-deficient and neutrophil-depleted mice. In addition, clinical E. coli strains that were more resistant to LL-37 caused more severe urinary tract infections than did susceptible strains. Thus, cathelicidin seems to be a key factor in mucosal immunity of the urinary tract.

L12 ANSWER 4 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:413151 BIOSIS
DN PREV200600417245
TI Cathelicidin deficiency predisposes to eczema herpeticum.
AU Howell, Michael D.; Wollenberg, Andreas; Gallo, Richard L.; Flaig, Michael; Streib, Joanne E.; Wong, Cathy; Pavicic, Tatjana; Boguniewicz, Mark; Leung, Donald Y. M. [Reprint Author]
CS Natl Jewish Med and Res Ctr, Dept Pediat, Div Allergy and Immunol, 1400 Jackson St,Room K926I, Denver, CO 80206 USA
leungd@njc.org
SO Journal of Allergy and Clinical Immunology, (APR 2006) Vol. 117, No. 4, pp. 836-841.
CODEN: JACIBY. ISSN: 0091-6749.
DT Article
LA English
ED Entered STN: 23 Aug 2006
Last Updated on STN: 23 Aug 2006
AB Background: The cathelicidin family of antimicrobial peptides is an integral component of the innate immune response that exhibits activity against bacterial, fungal, and viral pathogens. Eczema herpeticum (ADEH) develops in a subset of patients with atopic dermatitis (AD) because of disseminated infection with herpes simplex virus (HSV).Objective: This study investigated the potential role of cathelicidins in host susceptibility to HSV infection .Methods: Glycoprotein D was measured by means of real-time RT-PCR as a

marker of HSV replication in skin biopsy specimens and human keratinocyte cultures. Cathelicidin expression was evaluated in skin biopsy specimens from patients with AD (n = 10) without a history of HSV skin infection and from patients with ADEH (n = 10).Results: The cathelicidin peptide LL-37 (human cathelicidin) exhibited activity against HSV in an antiviral assay, with significant killing (P <.001) within the physiologic range. The importance of cathelicidins in antiviral skin host defense was confirmed by the observation of higher levels of HSV-2 replication in cathelicidin-deficient (Cnlp(-/-)) mouse skin (2.6 +/- 0.5 pg HSV/pg GAPDH, P <.05) compared with that seen in skin from their wild-type counterparts (0.9 +/- 0.3). Skin from patients with ADEH exhibited significantly (P <.05) lower levels of cathelicidin protein expression than skin from patients with AD. We also found a significant inverse correlation between cathelicidin expression and serum IgE levels (r(2) = 0.46, P <.05) in patients with AD and patients with ADEH.Conclusion: This study demonstrates that the cathelicidin peptide LL-37 possesses antiviral activity against HSV and demonstrates the importance of variable skin expression of cathelicidins in controlling susceptibility to ADEH. Additionally, serum IgE levels might be a surrogate marker for innate immune function and serve as a biomarker for which patients with AD are susceptible to ADEH.Clinical implications: A deficiency of LL-37 might render patients with AD susceptible to ADEH. Therefore increasing production of skin LL-37 might prevent herpes infection in patients with AD.

L12 ANSWER 5 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:305375 BIOSIS
DN PREV200600300340
TI The mammalian ionic environment dictates microbial susceptibility to antimicrobial defense peptides.
AU Dorschner, Robert A.; Lopez-Garcia, Belen; Peschel, Andreas; Kraus, Dirk; Morikawa, Kazuya; Nizet, Victor; Gallo, Richard L. [Reprint Author]
CS Mail Code 9111B,3350 La Jolla Viallage Dr, La Jolla, CA 92161 USA
rgallo@vapop.ucsd.edu
SO FASEB Journal, (JAN 2006) Vol. 20, No. 1, pp. 35-42.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Article
LA English
ED Entered STN: 7 Jun 2006
Last Updated on STN: 7 Jun 2006
AB Antimicrobial peptides (AMPs) have been shown in animal and human systems to be effective natural antibiotics. However, it is unclear how they convey protection; they often appear inactive when assayed under culture conditions applied to synthetic antibiotics. This inactivation has been associated with loss of function in physiological concentrations of NaCl or serum. In this study we show that the balance of host ionic conditions dictate microbial sensitivity to AMPs. Carbonate is identified as the critical ionic factor present in mammalian tissues that imparts the ability of AMPs such as cathelicidins and defensins to kill at physiological NaCl concentrations. After adapting to carbonate-containing solutions, global changes occur in Staphylococcus aureus and Escherichia coli structure and gene expression despite no change in growth rate. Our findings show that changes in cell wall thickness and Sigma factor B expression correspond to the increased susceptibility to the AMP LL-37. These observations provide new insight into the factors involved in enabling function of innate immune effector molecules, and suggest that discovery of new antimicrobials should specifically target pathogens as they exist in the host and not the distinctly different phenotype of bacteria grown in culture broth.-Dorschner, R. A., Lopez-Garcia, B., Peschel, A., Kraus, D., Morikawa, K., Nizet, V., Gallo, R. L. The mammalian ionic environment dictates microbial susceptibility to antimicrobial defense peptides.

L12 ANSWER 6 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:550460 BIOSIS
DN PREV200510341732
TI 7h2 cytokines down-regulate cathelicidin expression and increase skin susceptibility to viral infection in atopic dermatitis (AD) patients.
AU Howell, Michael D. [Reprint Author]; Boguniewicz, Mark; Streib, Joanne E.; Wong, Cathy; Gallo, Richard L.; Leung, Donald Y. M.
CS Natl Jewish Med and Res Ctr, Denver, CO USA
howellm@njc.org
SO Journal of Investigative Dermatology, (SEP 2005) Vol. 125, No. 3, pp. 597. Meeting Info.: 4th Georg Rajka International Symposium on Atopic Dermatitis. Arcachon, FRANCE. September 15 -17, 2005.
CODEN: JIDEAE. ISSN: 0022-202X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 7 Dec 2005
Last Updated on STN: 7 Dec 2005

L12 ANSWER 7 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:514686 BIOSIS
DN PREV200510305266
TI Expression and modulation of LL-37 in normal human keratinocytes, HaCaT cells, and inflammatory skin diseases.
AU Kim, Ji Eun; Kim, Beom Joon; Jeong, Mi Sook; Seo, Seong Jun [Reprint Author]; Kim, Myeung Nam; Hong, Chang Kwun; Ro, Byung In
CS Chung Ang Univ, Med Ctr, Dept Dermatol, Coll Med, 224-1 Heukseok Dong, Seoul 156756, South Korea
drseo@hanafos.com
SO Journal of Korean Medical Science, (AUG 2005) Vol. 20, No. 4, pp. 649-654.
CODEN: JKMSEH. ISSN: 1011-8934.
DT Article
LA English
ED Entered STN: 23 Nov 2005
Last Updated on STN: 23 Nov 2005
AB Defensins and cathelicidins (LL-37) are major antimicrobial peptides (AMPs) of the innate immune system of the human skin. In normal non-inflamed skin these peptides are negligible, but their expression can be markedly increased in inflammatory skin disease such as psoriasis. We designed this study to identify the expressions of LL-37 in normal human keratinocyte (NHK) and HaCaT cells after exposure to stimulants and to investigate difference of LL -37 expression accompanied with cell differentiation status, and come to understand difference of susceptibility to infection in atopic dermatitis and psoriasis. Expressions of LL-37 in NHKs and HaCaT cells were evaluated by using RT-PCR, Western blotting, and immunohistochemical (IHC) staining at 6, 12, and 24 hr post stimulation after exposure to Ultraviolet B irradiation and lipopolysaccharide. And expression of LL-37 in skin biopsy specimens from patients with atopic dermatitis and psoriasis was determined by immunohistochemical analysis. In time-sequential analyses of LL-37 expression revealed that LL-37 was expressed in NHKs, but not in HaCaT cells. IHC analysis confirmed the presence of abundant LL-37 in the epidermis of psoriasis. Therefore we deduced that expression of LL-37 is affected by UV irradiation, bacterial infection, and status of cell differentiation.

L12 ANSWER 8 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:423615 BIOSIS
DN PREV200510206262
TI Cationic antimicrobial peptide resistance in Neisseria meningitidis.
AU Tzeng, Yih-Ling; Ambrose, Karita D.; Zughaier, Susu; Zhou, Xiaoliu;

Miller, Yoon K.; Shafer, William M.; Stephens, David S. [Reprint Author]
CS Dept Vet Affairs Med Ctr, Lab Bacterial Pathogenesis, Res 151,Room 5A188,1670 Clairmont Rd, Atlanta, GA 30033 USA
dstep01@emory.edu
SO Journal of Bacteriology, (AUG 2005) Vol. 187, No. 15, pp. 5387-5396.
CODEN: JOBAAY. ISSN: 0021-9193.
DT Article
LA English
ED Entered STN: 19 Oct 2005
Last Updated on STN: 19 Oct 2005
AB Cationic antimicrobial peptides (CAMPs) are important components of the innate host defense system against microbial infections and microbial products. However, the human pathogen Neisseria meningitidis is intrinsically highly resistant to CAMPs, such as polymyxin B (PxB) (MIC >= 512 mu g/ml). To ascertain the mechanisms by which meningococci resist PxB, mutants that displayed increased sensitivity (>= 4-fold) to PxB were identified from a library of mariner transposon mutants generated in a meningococcal strain, NMB. Surprisingly, more than half of the initial PxB-sensitive mutants had insertions within the mtrCDE operon, which encodes proteins forming a multidrug efflux pump. Additional PxB-sensitive mariner mutants were identified from a second round of transposon mutagenesis performed in an mtr efflux pump-deficient background. Further, a mutation in lptA, the phosphoethanolamine (PEA) transferase responsible for modification of the lipid A head groups, was identified to cause the highest sensitivity to PxB. Mutations within the mtrD or lptA genes also increased meningococcal susceptibility to two structurally unrelated CAMPs, human LL-37 and protegrin-1. Consistently, PxB neutralized inflammatory responses elicited by the lptA mutant lipooligosaccharide more efficiently than those induced by wild-type lipooligosaccharide. mariner mutants with increased resistance to PxB were also identified in NMB background and found to contain insertions within the pilMNOPQ operon involved in pilin biogenesis. Taken together, these data indicated that meningococci utilize multiple mechanisms including the action of the MtrC-MtrD-MtrE efflux pump and lipid A modification as well as the type IV pilin secretion system to modulate levels of CAMP resistance. The modification of meningococcal lipid A head groups with PEA also prevents neutralization of the biological effects of endotoxin by CAMP.

L12 ANSWER 9 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:319348 BIOSIS
DN PREV200510114743
TI Skin microenvironment dictates susceptibility of bacteria to antimicrobial peptides.
AU Dorschner, R. A. [Reprint Author]; Lopez-Garcia, B.; Gallo, R. L.
CS Univ Calif San Diego, San Diego, CA 92103 USA
SO Journal of Investigative Dermatology, (MAR 2004) Vol. 122, No. 3, pp. A133,A132.
Meeting Info.: 65th Annual Meeting of the Society-for-Investigative-Dermatology. Providence, RI, USA. April 28 -May 01, 2004. Soc Investigat Dermatol.
CODEN: JIDEAE. ISSN: 0022-202X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Aug 2005
Last Updated on STN: 25 Aug 2005

L12 ANSWER 10 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:164925 BIOSIS
DN PREV200500164248
TI Marked reduction of LL-37/hCAP-18, an antimicrobial peptide, in patients with acute myeloid leukemia.

AU An, Li-Li; Ma, Xiao-Tong; Yang, Ying-Hua; Lin, Yon-Min; Son, Yu-Hua; Wu, Ke-Fu [Reprint Author]
CS Inst HematolNatl Lab Expt Hematol, Chinese Acad Med Sci, 288 Nanjing Rd, Tianjin, 300020, China
kfwu@public.tpt.tj.cn
SO International Journal of Hematology, (January 2005) Vol. 81, No. 1, pp. 45-47. print.
ISSN: 0925-5710 (ISSN print).
DT Article
LA English
ED Entered STN: 27 Apr 2005
Last Updated on STN: 27 Apr 2005
AB We detected LL-37/hCAP-18 expression in the peripheral blood smears of 50 healthy donors and 143 patients with various hematological diseases. Compared with that in the healthy donors. expression of the protein in the neutrophils was significantly lower in patients with acute myeloid leukemia (AML), especially those with infection. but no significant difference was detected in messenger RNA level. We did not detect increased LL-37/hCAP-18 protein expression in U937 cells treated with lipopolysaccharide or Staphylococcus aureus Cowan strain. Furthermore, LL-37 /hCAP-18 protein production was not restored in differentiated myeloid cell lines NB4 or HL-60 induced by all-trans retinoic acid. LL-37/hCAP-18 has been;shown to play, a role in host defense. and its deficiency in AML may be one of the explanations for susceptibility to infection among. these patients.

L12 ANSWER 11 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:162814 BIOSIS
DN PREV200500162485
TI Expression and activity of beta-defensins and LL-37 in the developing human lung.
AU Starner, Timothy D. [Reprint Author]; Agerberth, Birgitta; Gudmundsson, Gudmundur H.; McCray, Paul B. Jr
CS Dept Pediat, Univ Iowa, 2JCP,200 Hawkins Dr, Iowa City, IA, 52242, USA
timothy-starner@uiowa.edu
SO Journal of Immunology, (February 1 2005) Vol. 174, No. 3, pp. 1608-1615. print.
ISSN: 0022-1767 (ISSN print).
DT Article
LA English
ED Entered STN: 27 Apr 2005
Last Updated on STN: 27 Apr 2005
AB Immaturity of innate immunity contributes to the increased susceptibility of human neonates to infection. The lung is a major portal of entry for potential pathogens in the neonate, and human beta-defensins (HBDs) and LL-37 participate in pulmonary innate immunity. We hypothesized that these antimicrobial factors would be developmentally regulated, expressed by neonatal pulmonary tissues, and participate in neonatal innate immunity. We found HBD-2 to be the predominant beta-defensin in human neonatal lung. HBD-2 mRNA expression was developmentally regulated, induced by the proinflammatory factor, IL-1beta, and decreased by dexamethasone. Additionally, HBD-2 abundance in neonatal tracheal aspirates increased as a function of gestational age. HBD-1 had a lower level of expression compared with HBD-2 and was induced by dexamethasone. HBD-3 and LL-37 messages were not detected in airway epithelial cultures. Additionally, each antimicrobial peptide exhibited a unique spectrum of antimicrobial activity and salt sensitivity against bacteria commonly causing sepsis in the neonate. Lower levels of HBD-2 may be one factor contributing to the increased susceptibility of premature infants to pulmonary infections.

L12 ANSWER 12 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:69947 BIOSIS
DN PREV200500068901
TI Degradation of human antimicrobial peptide LL-37 by Staphylococcus aureus-derived proteinases.
AU Sieprawska-Lupa, Magdalena; Mydel, Piotr; Krawczyk, Katarzyna; Wojcik, Kinga; Puklo, Magdalena; Lupa, Boguslaw; Suder, Piotr; Silberring, Jerzy; Reed, Matthew; Pohl, Jan; Shafer, William; McAleese, Fionnuala; Foster, Timothy; Travis, Jim; Potempa, Jan [Reprint Author]
CS Dept Biochem and Mol Biol, Univ Georgia, Life Sci Bldg,E Green St, Athens, GA, 30602, USA
potempa@uga.edu
SO Antimicrobial Agents and Chemotherapy, (December 2004) Vol. 48, No. 12, pp. 4673-4679. print.
ISSN: 0066-4804 (ISSN print).
DT Article
LA English
ED Entered STN: 16 Feb 2005
Last Updated on STN: 16 Feb 2005
AB Cathelicidin LL-37 is one of the few human bactericidal peptides with potent antistaphylococcal activity. In this study we examined the susceptibility of LL-37 to proteolytic degradation by two major proteinases produced by Staphylococcus aureus, a metalloproteinase (aureolysin) and a glutamylendopeptidase (V8 protease). We found that aureolysin cleaved and inactivated LL-37 in a time- and concentration-dependent manner. Analysis of the generated fragments by mass spectroscopy revealed that the initial cleavage of LL-37 by aureolysin occurred between the Arg19-Ile20, Arg23-Ile24, and Leu31-Val32 peptide bonds, instantly annihilating the antibacterial activity of LL-37. In contrast, the V8 proteinase hydrolyzed efficiently only the Glu16-Phe17 peptide bond, rendering the C-terminal fragment refractory to further degradation. This fragment (termed LL-17-37) displayed antibacterial activity against S. aureus at a molar level similar to that of the full-length LL-37 peptide, indicating that the antibacterial activity of LL-37 resides in the C-terminal region. In keeping with LL-37 degradation by aureolysin, S. aureus strains that produce significant amounts of this metalloprotease were found to be less susceptible to LL-17-37 than strains expressing no aureolysin activity. Taken together, these data suggest that aureolysin production by S. aureus contributes to the resistance of this pathogen to the innate immune system of humans mediated by LL-37.

L12 ANSWER 13 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:176782 BIOSIS
DN PREV200400177086
TI Increased propensity of atopic dermatitis (AD) patients to viral infections may be due to Th2 cytokines which down-regulate cathelicidins with anti-viral activity.
AU Howell, M. D. [Reprint Author]; Streib, J. E. [Reprint Author]; Kisich, K. O. [Reprint Author]; Jones, J. F.; Gallo, R. L.; Leung, D. Y. M. [Reprint Author]
CS Department of Pediatrics, National Jewish Medical and Research Center, Denver, CO, USA
SO Journal of Allergy and Clinical Immunology, (February 2004) Vol. 113, No. 2 Supplement, pp. S52. print.
Meeting Info.: 60th Annual Meeting of the American Academy of Allergy, Asthma and Immunology (AAAAI). San Francisco, CA, USA. March 19-23, 2004. American Academy of Allergy, Asthma and Immunology.
CODEN: JACIBY. ISSN: 0091-6749.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 31 Mar 2004
Last Updated on STN: 31 Mar 2004

L12 ANSWER 14 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:173219 BIOSIS
DN PREV200400174632
TI Selective killing of vaccinia virus by LL-37: Implications for eczema vaccinatum.
AU Howell, Michael D.; Jones, James F.; Kisich, Kevin O.; Streib, Joanne E.; Gallo, Richard L.; Leung, Donald Y. M. [Reprint Author]
CS Department of Pediatrics, National Jewish Medical and Research Center, 1400 Jackson Street, Room K926, Denver, CO, 80206, USA
leungd@njc.org
SO Journal of Immunology, (February 1 2004) Vol. 172, No. 3, pp. 1763-1767. print.
ISSN: 0022-1767 (ISSN print).
DT Article
LA English
ED Entered STN: 31 Mar 2004
Last Updated on STN: 31 Mar 2004
AB Possible bioterrorism with smallpox has led to the resumption of smallpox (vaccinia virus) immunization. One complication, eczema vaccinatum, occurs primarily in patients with atopic dermatitis (AD). Skin lesions of patients with AD, but not psoriasis, is deficient in the cathelicidin antimicrobial peptide (LL-37) and human beta-defensin-2 (HBD-2). We hypothesized that this defect may explain the susceptibility of patients with AD to eczema vaccinatum. The Wyeth vaccine strain of vaccinia virus was incubated with varying concentrations of human (LL-37) and murine (CRAMP) cathelicidins, human alpha-defensin (HBD-1, HBD-2), and a control peptide. Outcomes included quantification of viral PFU, vaccinia viral gene expression by quantitative real-time RT-PCR, and changes in virion structure by transmission electron microscopy. CRAMP knockout mice and control animals were inoculated by skin pricks with 2X105 PFU of vaccinia and examined daily for pox development. Physiologic amounts of human and murine cathelicidins (10-50 muM), but not human defensins, which had antibacterial activity, resulted in the in vitro reduction of vaccinia viral plaque formation ($p<0.0001$), vaccinia mRNA expression ($p<0.001$), and alteration of vaccinia virion structure. In vivo vaccinia pox formation occurred in four of six CRAMP knockout animals and in only one of 15 control mice ($p<0.01$). These data support a role for cathelicidins in the inhibition of orthopox virus (vaccinia) replication both in vitro and in vivo. Susceptibility of patients with AD to eczema vaccinatum may be due to a deficiency of cathelicidin.

L12 ANSWER 15 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:168227 BIOSIS
DN PREV200400170386
TI Polysaccharide intercellular adhesin (PIA) protects Staphylococcus epidermidis against major components of the human innate immune system.
AU Vuong, Cuong; Voyich, Jovanka M.; Fischer, Elizabeth R.; Braughton, Kevin R.; Whitney, Adeline R.; DeLeo, Frank R.; Otto, Michael [Reprint Author]
CS Laboratory of Human Bacterial Pathogenesis, Rocky Mountain Laboratories, National Institute of Allergy and Infectious Diseases, National Institutes of Health, 903 S. 4th Street, Hamilton, MT, 59840, USA
motto@niaid.nih.gov
SO Cellular Microbiology, (March 2004) Vol. 6, No. 3, pp. 269-275. print.
ISSN: 1462-5814 (ISSN print).
DT Article

LA English
ED Entered STN: 24 Mar 2004
Last Updated on STN: 24 Mar 2004
AB The skin commensal and opportunistic pathogen Staphylococcus epidermidis is the leading cause of nosocomial and biofilm-associated infections. Little is known about the mechanisms by which S. epidermidis protects itself against the innate human immune system during colonization and infection. We used scanning electron microscopy to demonstrate that the exopolysaccharide intercellular adhesin (PIA) resides in fibrous strands on the bacterial cell surface, and that lack of PIA production results in complete loss of the extracellular matrix material that has been suggested to mediate immune evasion. Phagocytosis and killing by human polymorphonuclear leucocytes was significantly increased in a mutant strain lacking PIA production compared with the wild-type strain. The mutant strain was also significantly more susceptible to killing by major antibacterial peptides of human skin, cationic human beta-defensin 3 and LL-37, and anionic dermcidin. PIA represents the first defined factor of the staphylococcal biofilm matrix that protects against major components of human innate host defence.

L12 ANSWER 16 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2003:510403 BIOSIS
DN PREV200300497238
TI Cationic antimicrobial peptides activate a two-component regulatory system, PmrA-PmrB, that regulates resistance to polymyxin B and cationic antimicrobial peptides in Pseudomonas aeruginosa.
AU McPhee, Joseph B.; Lewenza, Shawn; Hancock, Robert E. W. [Reprint Author]
CS Department of Microbiology and Immunology, University of British Columbia, 300-6174 University Blvd., Vancouver, BC, V6T 1Z3, Canada
bob@cmdr.ubc.ca
SO Molecular Microbiology, (October 2003) Vol. 50, No. 1, pp. 205-217. print.
ISSN: 0950-382X (ISSN print).
DT Article
LA English
ED Entered STN: 29 Oct 2003
Last Updated on STN: 29 Oct 2003
AB The two-component regulatory system PhoP-PhoQ of Pseudomonas aeruginosa regulates resistance to cationic antimicrobial peptides, polymyxin B and aminoglycosides in response to low Mg2+ conditions. We have identified a second two-component regulatory system, PmrA-PmrB, that regulates resistance to polymyxin B and cationic antimicrobial peptides. This system responds to limiting Mg2+, and is affected by a phoQ, but not a phoP mutation. Inactivation of the pmrB sensor kinase and pmrA response regulator greatly decreased the expression of the operon encoding pmrA-pmrB while expression of the response regulator pmrA in trans resulted in increased activation suggesting that the pmrA-pmrB operon is autoregulated. Interposon mutants in pmrB, pmrA, or in an intergenic region upstream of pmrA-pmrB exhibited two to 16-fold increased susceptibility to polymyxin B and cationic antimicrobial peptides. The pmrA-pmrB operon was also found to be activated by a number of cationic peptides including polymyxins B and E, cattle indolicidin and synthetic variants as well as LL-37, a component of human innate immunity, whereas peptides with the lowest minimum inhibitory concentrations tended to be the weakest inducers. Additionally, we showed that the putative LPS modification operon, PA3552-PA3559, was also induced by cationic peptides, but its expression was only partially dependent on the PmrA-PmrB system. The discovery that the PmrA-PmrB two-component system regulates resistance to cationic peptides and that both it and the putative LPS modification system are induced by cationic antimicrobial peptides has major implications for the development of these antibiotics as a therapy for P. aeruginosa infections.

L12 ANSWER 17 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2003:389472 BIOSIS
DN PREV200300389472
TI Expression of LL-37/hCAP-18 gene in human leukemia cells.
AU Yang, Ying-Hua; Zheng, Guo-Guang; Li, Ge; Zhang, Bin; Song, Yu-Hua; Wu, Ke-Fu [Reprint Author]
CS National Laboratory of Experimental Hematology, Institute of Hematology and Blood Diseases Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, 288 Nanjing Road, Tianjin, 300020, China kfwu@public.tpt.tj.cn
SO Leukemia Research, (October 2003) Vol. 27, No. 10, pp. 947-950. print. ISSN: 0145-2126 (ISSN print).
DT Article
LA English
ED Entered STN: 20 Aug 2003
Last Updated on STN: 20 Aug 2003
AB LL-37/hCAP-18 is an important part of host defense. Several diseases in human are characterized by impairment in the function of LL-37/hCAP-18 peptide. We examined the expression of LL-37/hCAP-18 in a panel of hematopoietic cell lines representing multiple cell lineages. LL-37 /hCAP-18 expression at mRNA level was detected and varied among six of nine cell lines. The level of Raji cells was about eight folds higher than that of Ramos cells. However, only two cell lines, J6-1 and U937, expressed protein products. We also investigated LL-37 /hCAP-18 protein in nine leukemia and three idiopathic thrombocytopenic purpura (ITP) patients via immunocytochemical staining. The rate of LL-37/hCAP-18 positive cells ranged from 60 (ITP) to 0.5% (M5). These data suggested that the low translation efficiency of LL-37/hCAP-18 expresses in some leukemia cells might be one of the reasons that leukemia patients were susceptible to infection.

L12 ANSWER 18 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2003:215664 BIOSIS
DN PREV200300215664
TI Neonatal skin in mice and humans expresses increased levels of antimicrobial peptides: Innate immunity during development of the adaptive response.
AU Dorschner, Robert A.; Lin, Kenneth H.; Murakami, Masamoto; Gallo, Richard L. [Reprint Author]
CS 3350 LaJolla Village Drive, Mail Code 111B, San Diego, CA, 92161, USA rgallo@UCSD.edu
SO Pediatric Research, (April 2003) Vol. 53, No. 4, pp. 566-572. print. ISSN: 0031-3998 (ISSN print).
DT Article
LA English
ED Entered STN: 30 Apr 2003
Last Updated on STN: 30 Apr 2003
AB The expression of antimicrobial peptides and proteins is an important innate immune defense mechanism that has recently been shown to be essential for cutaneous defense against invasive bacterial disease. Newborns have an immature cellular immune defense system that leads to increased susceptibility to infections. Here we show that skin from embryonic and newborn mice, as well as human newborn foreskin, express antimicrobial peptides of the cathelicidin and beta-defensin gene families. Immunohistochemistry and in situ hybridization demonstrated abundant cathelicidin protein and mRNA is present in normal skin during the perinatal period. Quantitative real-time PCR showed mouse cathelicidin expression (CRAMP) is 10- to 100-fold greater in the perinatal period than adult. Murine

beta-defensins-1 and -4 and human beta-defensin-2 were also present in newborn skin. Combined, human cathelicidin (LL-37 /hCAP/18) and beta-defensin-2 demonstrated synergistic antimicrobial activity and efficiently killed group B Streptococcus, an important neonatal pathogen. Antimicrobial peptides may therefore provide a compensatory innate defense mechanism during development of cellular immune response mechanisms in the newborn period.

L12 ANSWER 19 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:552782 BIOSIS
DN PREV200200552782
TI Endogenous antimicrobial peptides and skin infections in atopic dermatitis.
AU Ong, Peck Y.; Ohtake, Takaaki; Brandt, Corinne; Strickland, Ian; Boguniewicz, Mark; Ganz, Tomas; Gallo, Richard L.; Leung, Donald Y. M. [Reprint author]
CS Department of Pediatrics, National Jewish Medical and Research Center, 1400 Jackson St., Rm. K926, Denver, CO, 80206, USA
SO New England Journal of Medicine, (October 10, 2002) Vol. 347, No. 15, pp. 1151-1160. print.
CODEN: NEJMAG. ISSN: 0028-4793.
DT Article
LA English
ED Entered STN: 30 Oct 2002
Last Updated on STN: 30 Oct 2002
AB Background: The innate immune system of human skin contains antimicrobial peptides known as cathe-licidins (LL-37) and beta-defensins. In normal skin these peptides are negligible, but they accumulate in skin affected by inflammatory diseases such as psoriasis. We compared the levels of expression of LL-37 and human beta-defensin 2 (HBD-2) in inflamed skin from patients with atopic dermatitis and from those with psoriasis. Methods: The expression of LL-37 and HBD-2 protein in skin-biopsy specimens from patients with psoriasis, patients with atopic dermatitis, and normal subjects was determined by immunohistochemical analysis. The amount of antimicrobial peptides in extracts of skin samples was also analyzed by immunodot blot analysis (for LL-37) and Western blot analysis (for HBD-2). Quantitative, real-time reverse-transcriptase-polymerase-chain-reaction (RT-PCR) assays were used to confirm the relative expression of HBD-2 and LL-37 messenger RNA (mRNA) in the skin-biopsy specimens. These peptides were also tested for antimicrobial activity against Staphylococcus aureus with the use of a colony-forming assay. Results: Immunohistochemical analysis confirmed the presence of abundant LL-37 and HBD-2 in the superficial epidermis of all patients with psoriasis. In comparison, immunostaining for these peptides was significantly decreased in acute and chronic lesions from patients with atopic dermatitis (P=0.006 and P=0.03, respectively). These results were confirmed by immunodot blot and Western blot analyses. Real-time RT-PCR showed significantly lower expression of HBD-2 mRNA and LL-37 mRNA in atopic lesions than in psoriatic lesions (P=0.009 and P=0.02, respectively). The combination of LL-37 and HBD-2 showed synergistic antimicrobial activity by effectively killing S. aureus. Conclusions: A deficiency in the expression of antimicrobial peptides may account for the susceptibility of patients with atopic dermatitis to skin infection with S. aureus.

L12 ANSWER 20 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2002:304079 BIOSIS
DN PREV200200304079
TI Borrelia burgdorferi are susceptible to killing by a variety of human polymorphonuclear leukocyte components.

AU Lusitani, Denise; Malawista, Stephen E.; Montgomery, Ruth R. [Reprint author]
CS Dept. of Internal Medicine, Yale University School of Medicine, 333 Cedar St., LCI 608, New Haven, CT, 06520-8031, USA
ruth.montgomery@yale.edu
SO Journal of Infectious Diseases, (15 March, 2002) Vol. 185, No. 6, pp. 797-804. print.
CODEN: JIDIAQ. ISSN: 0022-1899.
DT Article
LA English
ED Entered STN: 22 May 2002
Last Updated on STN: 22 May 2002
AB The killing of Borrelia burgdorferi by intact human polymorphonuclear leukocytes (PMNL) and by individual PMNL components was compared. Intact PMNL killed B. burgdorferi 6.5-fold more efficiently and 5-fold more completely when spirochetes were opsonized with specific antibodies. U-cytoplasts, which have activatable oxidase, killed opsonized B. burgdorferi with an efficiency similar to that of intact PMNL in killing unopsonized B. burgdorferi. Although B. burgdorferi were susceptible to H2O2 and nitric oxide, PMNL lysates killed B. burgdorferi nearly as well as intact PMNL killed opsonized B. burgdorferi, suggesting a critical role for granule contents. B. burgdorferi were killed by the PMNL antimicrobial components elastase, LL-37, bactericidal/permeability-increasing protein, and human neutrophil peptide-1. B. burgdorferi had limited susceptibility to killing by lysozyme and were not killed by azurocidin, proteinase 3, or lactoferrin. The efficient killing of B. burgdorferi by a variety of PMNL mechanisms highlights the paradoxical persistence of spirochetes in vivo.

L12 ANSWER 21 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2000:405181 BIOSIS
DN PREV200000405181
TI Activity of cecropin P1 and FA-LL-37 against urogenital microflora.
AU Smeianov, Vladimir [Reprint author]; Scott, Kellie; Reid, Gregor
CS Lawson Research Institute, University of Western Ontario, London, ON, Canada
SO Microbes and Infection, (June, 2000) Vol. 2, No. 7, pp. 773-777. print.
ISSN: 1286-4579.
DT Article
LA English
ED Entered STN: 20 Sep 2000
Last Updated on STN: 8 Jan 2002
AB Two mammalian antimicrobial peptides, FA-LL-37 and cecropin P1, were tested for activity against six uropathogens and five Lactobacillus strains by broth microdilution assay. Both peptides inhibited Escherichia coli at 25 muM (FA-LL-39), and 1.56 muM (cecropin P1), Pseudomonas aeruginosa (12.5 muM, and 25 muM), and Klebsiella pneumoniae, (50 muM, and 1.56 muM), but not Enterococcus faecalis and Staphylococcus epidermidis. FA-LL-37 acted bacteriocidally against E. coli and bacteriostatically against the other two Gram-negative organisms. Cecropin P1 was bacteriocidal to all susceptible bacteria. Lactobacilli were resistant to both peptides, with the exception of poultry isolate Lactobacillus fermentum B-54, which was susceptible to FA-LL-37 at 100 muM. The differential activities of these peptides toward Gram-negative uropathogens versus urogenital lactobacilli demonstrate their potential as a topical treatment for urinary tract infections. In addition, production of such peptides in vivo could be a natural mechanism to aid in the maintenance of the lactobacilli-dominated urogenital flora at the expense of pathogens.

L12 ANSWER 22 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN
AN 1998:444891 BIOSIS
DN PREV199800444891
TI Activities of LL-37, a cathelin-associated antimicrobial peptide of human neutrophils.
AU Turner, Jeffrey; Cho, Yoon; Dinh, Nhu-Nguyen; Waring, Alan J.; Lehrer, Robert I. [Reprint author]
CS Dep. Med., Cent. Health Sci., Box 951690, Los Angeles, CA 90095-1690, USA
SO Antimicrobial Agents and Chemotherapy, (Sept., 1998) Vol. 42, No. 9, pp. 2206-2214. print.
CODEN: AMACCQ. ISSN: 0066-4804.
DT Article
LA English
ED Entered STN: 21 Oct 1998
Last Updated on STN: 21 Oct 1998
AB Human neutrophils contain two structurally distinct types of antimicrobial peptides, beta-sheet defensins (HNP-1 to HNP-4) and the alpha-helical peptide LL-37. We used radial diffusion assays and an improved National Committee for Clinical Laboratory Standards-type broth microdilution assay to compare the antimicrobial properties of LL-37, HNP-1, and protegrin (PG-1). Although generally less potent than PG-1, LL-37 showed considerable activity (MIC, <10 mug/ml) against Pseudomonas aeruginosa, Salmonella typhimurium, Escherichia coli, Listeria monocytogenes, Staphylococcus epidermidis, Staphylococcus aureus, and vancomycin-resistant enterococci, even in media that contained 100 mM NaCl. Certain organisms (methicillin-resistant S. aureus, Proteus mirabilis, and Candida albicans) were resistant to LL-37 in media that contained 100 mM NaCl but were susceptible in low-salt media. Burkholderia cepacia was resistant to LL-37, PG-1, and HNP-1 in low- or high-salt media. LL-37 caused outer and inner membrane permeabilization of E. coli ML-35p. Chromogenic Limulus assays revealed that LL-37 bound to E. coli O111:B4 lipopolysaccharide (LPS) with a high affinity and that this binding showed positive cooperativity (Hill coefficient = 2.02). Circular dichroism spectrometry disclosed that LL-37 underwent conformational change in the presence of lipid A, transitioning from a random coil to an et-helical structure. The broad-spectrum antimicrobial properties of LL-37, its presence in neutrophils, and its inducibility in keratinocytes all suggest that this peptide and its precursor (hCAP-18) may protect skin and other tissues from bacterial intrusions and LPS-induced toxicity. The potent activity of LL-37 against P. aeruginosa, including mucoid and antibiotic-resistant strains, suggests that it or related molecules might have utility as topical bronchopulmonary microbicides in cystic fibrosis.

L12 ANSWER 23 OF 84 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 1998:167516 BIOSIS
DN PREV199800167516
TI Modulation of Neisseria gonorrhoeae susceptibility to vertebrate antibacterial peptide due to a member of the resistance/nodulation/divisio n efflux pump family.
AU Shafer, W. M. [Reprint author]; Qu, X.-D.; Waring, A. J.; Lehrer, R. I.
CS Dep. Microbiol. Immunol., Emory Univ. Sch. Med., Atlanta, GA 30322, USA
SO Proceedings of the National Academy of Sciences of the United States of America, (Feb. 17, 1998) Vol. 95, No. 4, pp. 1829-1833. print.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 6 Apr 1998
Last Updated on STN: 6 Apr 1998
AB We have previously described the antibacterial capacity of protegrin-1 (PG-1), a cysteine-rich, cationic peptide from porcine leukocytes, against

Neisseria gonorrhoeae. We now report genetic and biochemical evidence that gonococcal susceptibility to the lethal action of PG-1 and other structurally unrelated antibacterial peptides, including a peptide (LL-37) that is expressed constitutively by human granulocytes and testis and inducibly by keratinocytes, is modulated by an energy-dependent efflux system termed mtr. These results indicate that such efflux systems may enable mucosal pathogens like gonococci to resist endogenous antimicrobial peptides that are thought to act during infection.

L12 ANSWER 24 OF 84 CABA COPYRIGHT 2007 CABI on STN
AN 2005:125868 CABA
DN 20053117291
TI Anti-fungal activity of cathelicidins and their potential role in Candida albicans skin infection
AU Lopez-Garcia, B.; Lee, P. H. A.; Yamasaki, K.; Gallo, R. L.
CS Division of Dermatology, University of California San Diego, San Diego, CA 92161, USA. rgallo@vapop.ucsd.edu
SO Journal of Investigative Dermatology, (2005) Vol. 125, No. 1, pp. 108-115. 43 ref.
Publisher: Blackwell Publishing. Boston
ISSN: 0022-202X
URL: http://www.blackwell-synergy.com/servlet/useragent?func=showIssues&code=jid
CY United States
DT Journal
LA English
ED Entered STN: 3 Aug 2005
Last Updated on STN: 3 Aug 2005
AB Cathelicidins have broad anti-microbial capacity and are important for host defense against skin infections by some bacterial and viral pathogens. This study investigated the activity of cathelicidins against Candida albicans. The human cathelicidin LL-37, and mouse cathelicidin mCRAMP, killed C. albicans, but this fungicidal activity was dependent on culture conditions. Evaluation of the fungal membrane by fluorescent dye penetration after incubation with cathelicidins correlated membrane permeabilization and inhibition of fungal growth. Anti-fungal assays carried out in an ionic environment that mimicked human sweat and with the processed forms of cathelicidin such as are present in sweat found that the cleavage of LL-37 to forms such as RK-31 conferred additional activity against C. albicans. C. albicans also induced an increase in the expression of cathelicidin in mouse skin, but this induction did not confer systemic or subcutaneous resistance as mCRAMP-deficient mice were not more susceptible to C. albicans in blood-killing assays or in an intradermal infection model. Therefore, cathelicidins appear active against C. albicans, but may be most effective as a superficial barrier to infection.

L12 ANSWER 25 OF 84 CABA COPYRIGHT 2007 CABI on STN
AN 2005:68980 CABA
DN 20053050294
TI Mammalian host defense peptides
Advances in Molecular and Cellular Microbiology 6
AU Devine, D. A.; Hancock, R. E. W.; Devine, D. A. [EDITOR]; Hancock, R. E. W. [EDITOR]
CS Division of Oral Biology, Dental Institute, University of Leeds, Leeds, UK.
SO Mammalian host defense peptides, (2004) pp. xi + 393. many ref.
Publisher: Cambridge University Press. Cambridge
ISBN: 0-521-82220-3
CY United Kingdom
DT Book
LA English
ED Entered STN: 6 May 2005

Last Updated on STN: 6 May 2005

AB This book presents significant recent advances on mammalian antimicrobial peptides. The 13 chapters contain an overview of antimicrobial peptides, the role of cationic antimicrobial peptides in the regulation of commensal and pathogenic microbial populations, multiple functions of antimicrobial peptides in host immunity, therapeutic potential and applications of innate immunity peptides, mammalian [beta]-defensins in mucosal defence, biology and expression of the human cathelicidin LL-37 , antimicrobial peptides of the alimentary tract of mammals, antimicrobial peptides which suppress microbial infections and sepsis in animal models, the influence of bacterial structure and physiology on the susceptibility to cationic antimicrobial peptides, the antifungal mechanisms of antimicrobial peptides, antimicrobial peptides from platelets in defence against cardiovascular infections, mechanisms of bacterial resistance to antimicrobial peptides and the roles of antimicrobial peptides in pulmonary defence.

L12 ANSWER 26 OF 84 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1262902 CAPLUS
DN 146:117954
TI Penicillin-binding protein 1a promotes resistance of group B streptococcus to antimicrobial peptides
AU Hamilton, Andrea; Popham, David L.; Carl, David J.; Lauth, Xavier; Nizet, Victor; Jones, Amanda L.
CS Department of Pediatrics, University of Washington, Seattle, WA, USA
SO Infection and Immunity (2006), 74(11), 6179-6187
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB Evasion of host immune defenses is critical for the progression of invasive infections caused by the leading neonatal pathogen, group B streptococcus (GBS). Upon characterizing the factors required for virulence in a neonatal rat sepsis model, the authors found that a surface-associated penicillin-binding protein (PBP1a), encoded by ponA, played an essential role in resistance of GBS to phagocytic clearance. To elucidate how PBP1a promotes resistance to innate immunity, the authors compared the susceptibility of wild-type GBS and an isogenic ponA mutant to the bactericidal components of human neutrophils. The isogenic strains were found to be equally capable of blocking complement activation on the bacterial surface and equally associated with phagocytes and susceptible to oxidative killing. In contrast, the ponA mutant was significantly more susceptible to killing by cationic antimicrobial peptides (AMPs) of the cathelicidin and defensin families, which are now recognized as integral components of innate host defense against invasive bacterial infection. These observations may help explain the sensitivity to phagocytic killing and attenuated virulence of the ponA mutant. This novel function for PBP1a in promoting resistance of GBS to AMP did not involve an alteration in bacterial surface charge or peptidoglycan crosslinking. While the peptidoglycan polymerization and crosslinking activity of PBPs are essential for bacterial survival, the authors' study is the first to identify a role for a PBP in resistance to host AMPs.
RE.CNT 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 27 OF 84 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:868571 CAPLUS
DN 145:394867
TI Immunomodulatory properties of defensins and cathelicidins
AU Bowdish, D. M. E.; Davidson, D. J.; Hancock, R. E. W.
CS Centre for Microbial Diseases and Immunity Research, University of British Columbia, Vancouver, BC, V6T 1Z4, Can.
SO Current Topics in Microbiology and Immunology (2006), 306(Antimicrobial

Peptides and Human Disease), 27-66
CODEN: CTMIA3; ISSN: 0070-217X
PB Springer GmbH
DT Journal; General Review
LA English
AB A review. Host defense peptides are a conserved component of the innate immune response in all complex life forms. In humans, the major classes of host defense peptides include the α- and β-defensins and the cathelicidin, hCAP-18/LL-37. These peptides are expressed in the granules of neutrophils and by a wide variety of tissue types. They have many roles in the immune response including both indirect and direct antimicrobial activity, the ability to act as chemokines as well as induce chemokine production leading to recruitment of leukocytes to the site of infection, the promotion of wound healing and an ability to modulate adaptive immunity. It appears that many of these properties are mediated though direct interaction of peptides with the cells of the innate immune response including monocytes, dendritic cells, T cells and epithelial cells. The importance of these peptides in immune responses has been demonstrated since animals defective in the expression of certain host defense peptides show greater susceptibility to bacterial infections. In the very few instances in which human patients have been demonstrated to have defective host defense peptide expression, these individuals suffer from frequent infections. Although studies of the immunomodulatory properties of these peptides are in their infancy, there is a growing body of evidence suggesting that the immunomodulatory properties of these small, naturally occurring mols. might be harnessed for development as novel therapeutic agents.
RE.CNT 170 THERE ARE 170 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 28 OF 84 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:995932 CAPLUS
DN 141:420404
TI Anti-viral activity of cationic peptides of the cathelicidin family including LL-37, homologs and variants thereof, and diagnosing atopic dermatitis by detecting LL-37 or its gene
IN Gallo, Richard L.; Leung, Donald Y. M.; Jones, James F.
PA The Regents of the University of California, USA; National Jewish Medical and Research Center
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004098536	A2	20041118	WO 2004-US6952	20040305
	WO 2004098536	A3	20050616		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2006292551	A1	20061228	US 2006-546739	20060731
PRAI	US 2003-452906P	P	20030306		
	WO 2004-US6952	W	20040305		

AB The disclosure provides methods and compns. useful in the treatment of dermatitis and viral infections, and for determining predisposition to dermatitis. It was demonstrated by inventors that susceptibility of patients with atopic dermatitis (AD) to eczema vaccinatum may be due to a deficiency of cathelicidin. It was shown that physiol. amts. of human (LL-37) and murine (CRAMP) cathelicidins, but not human defensins, which had antibacterial activity, resulted in the in vitro reduction of vaccinia viral plaque formation, vaccinia mRNA expression, and alteration of vaccinia virion structure. The therapeutic compns. of invention comprise cationic peptides of the cathelicidin family including LL-37, CRAMP related homologs, and variants thereof. The disclosure further provides a LL-37 or CRAMP knock-out transgenic animal having increased susceptibility to viral infections of the skin, and useful for drug screening.

L12 ANSWER 29 OF 84 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:333962 CAPLUS
DN 140:353243
TI Method of diagnosis
IN Boman, Hans; Andersson, Mats; Puetsep, Katrin; Carlsson, Goeran
PA Mabtech Ab, Swed.
SO PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004034061	A2	20040422	WO 2003-EP11240	20031010
	WO 2004034061	A3	20040521		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2501926	A1	20040422	CA 2003-2501926	20031010
	AU 2003282027	A1	20040504	AU 2003-282027	20031010
	EP 1549957	A2	20050706	EP 2003-773640	20031010
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	JP 2006502395	T	20060119	JP 2004-542487	20031010
	US 2007059691	A1	20070315	US 2006-530606	20060221
PRAI	GB 2002-23655	A	20021010		
	WO 2003-EP11240	W	20031010		

AB A method is disclosed for determining the susceptibility of a subject to infection, which method comprises: (i) providing a sample from said subject; (ii) detecting any LL-37 present in said sample; (iii) optionally comparing the level of LL-37 in said sample to a control sample; and (iv) determining the susceptibility of said subject to infection, wherein no LL-37 or a low level of LL-37 indicates that said subject is susceptible to infection .

L12 ANSWER 30 OF 84 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
AN 2002026516 EMBASE
TI Interleukin-1α and interleukin-6 enhance the antibacterial properties of cultured composite keratinocyte grafts.

AU Erdag G.; Morgan J.R.
CS Dr. J.R. Morgan, Shriners Hospital for Children, 51 Blossom St., Boston, MA 02114, United States. jmorgan@sbi.org
SO Annals of Surgery, (2002) Vol. 235, No. 1, pp. 113-124. .
Refs: 39
ISSN: 0003-4932 CODEN: ANSUA5
CY United States
DT Journal; Article
FS 004 Microbiology
009 Surgery
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
ED Entered STN: 31 Jan 2002
Last Updated on STN: 31 Jan 2002
AB Objective: To determine whether the antibacterial properties of cultured composite keratinocyte grafts can be enhanced by cytokines that stimulate the innate immune response. Summary Background Data: Use of composite grafts of cultured keratinocytes has been limited because of their susceptibility to burn wound microorganisms as a result of their lack of a vasculature and immune cells when transplanted. Moreover, use of topical antimicrobial agents is limited with these composite grafts because of cytotoxic effects. Keratinocytes, like all epithelial cells in the body, maintain a natural defense mechanism called the innate immune system. Some components of this system can be induced by cytokines. Methods: The innate immune response of cultured composite keratinocyte grafts treated with various cytokines was assessed indirectly by measuring the levels of mRNA encoding antimicrobial peptides (human beta defensin-1 and -2, LL-37, and antileukoprotease) and antimicrobial proteins (lysozyme, bactericidal/permeability-inducing protein, and phospholipase A2) by reverse transcription-polymerase chain reaction and directly by measuring the ability of keratinocytes to inhibit the growth of added bacteria (Escherichia coli, Pseudomonas aeruginosa, and Staphylococcus aureus). Results: Treatment with interluekin-1α increased mRNA levels of antimicrobial peptides in keratinocytes on plastic dishes and in composite grafts. Interleukin-6 increased mRNA levels of antimicrobial proteins in composite grafts only. When added to composite grafts, both cytokines increased antibacterial activity against E. coli, P. aeruginosa, and S. aureus. Moreover, interleukin-1α and interleukin-6 did not impair the formation of a differentiated epidermis in vitro or after transplantation of the composite grafts. Conclusions: Treatment with interleukin-1α or interleukin-6 of cultured composite keratinocyte grafts stimulates the innate immune response of keratinocytes, enhances the antibacterial properties of these grafts, and may better prepare them to combat infections in contaminated burn wounds.

L12 ANSWER 31 OF 84 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:1171637 SCISEARCH
GA The Genuine Article (R) Number: 109KX
TI Induction of the antimicrobial peptide CRAMP in the blood-brain barrier and meninges after meningococcal infection
AU Bergman P; Johansson L; Wan H; Jones A; Gallo R L; Gudmundsson G H; Hokfelt T; Jonsson A B; Agerberth B (Reprint)
CS Karolinska Inst, Dept Med Biochem & Biophys, Scheeles Vag 2, SE-17177 Stockholm, Sweden (Reprint); Karolinska Inst, Dept Med Biochem & Biophys, SE-17177 Stockholm, Sweden; Karolinska Univ Hosp Huddinge, Dept Med, Ctr Infect Med, SE-14186 Stockholm, Sweden; Uppsala Univ, Biomed Ctr, Dept Med Biochem & Microbiol, SE-75123 Uppsala, Sweden; Univ Calif San Diego, Div Dermatol, La Jolla, CA 92161 USA; Vet Affairs San Diego Healthcare Ctr, La Jolla, CA 92161 USA; Univ Iceland, Inst Biol, IS-101 Reykjavik, Iceland; Karolinska Inst, Dept Neurosci, S-10401 Stockholm, Sweden

birgitta.agerberth@ki.se
CYA Sweden; USA; Iceland
SO INFECTION AND IMMUNITY, (DEC 2006) Vol. 74, No. 12, pp. 6982-6991.
ISSN: 0019-9567.
PB AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA.
DT Article; Journal
LA English
REC Reference Count: 45
ED Entered STN: 14 Dec 2006
Last Updated on STN: 14 Dec 2006
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Antimicrobial peptides are present in most living species and constitute important effector molecules of innate immunity. Recently, we and others have detected antimicrobial peptides in the brain. This is an organ that is rarely infected, which has mainly been ascribed to the protective functions of the blood-brain barrier (BBB) and meninges. Since the bactericidal properties of the BBB and meninges are not known, we hypothesized that antimicrobial peptides could play a role in these barriers. We addressed this hypothesis by infecting mice with the neuropathogenic bacterium Neisseria meningitidis. Brains were analyzed for expression of the antimicrobial peptide CRAMP by immunohistochemistry in combination with confocal microscopy. After infection, we observed induction of CRAMP in endothelial cells of the BBB and in cells of the meninges. To explore the functional role of CRAMP in meningococcal disease, we infected mice deficient of the CRAMP gene. Even though CRAMP did not appear to protect the brain from invasion of meningococci, CRAMP knockout mice were more susceptible to meningococcal infection than wild-type mice and exhibited increased meningococcal growth in blood, liver, and spleen. Moreover, we could demonstrate that carbonate, a compound that accumulates in the circulation during metabolic acidosis, makes meningococci more susceptible to CRAMP.

L12 ANSWER 32 OF 84 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:164390 SCISEARCH
GA The Genuine Article (R) Number: 009EO
TI Human alpha-defensins block papillomavirus infection
AU Buck C B; Day P M; Thompson C D; Lubkowski J; Lu W Y; Lowy D R; Schiller J T (Reprint)
CS NCI, Cellular Oncol Lab, Canc Res Ctr, Bethesda, MD 20892 USA (Reprint); NCI, Macromol Assembly Struct & Cell Signaling Sect, Ft Detrick, MD 21702 USA; Univ Maryland, Inst Biotechnol, Inst Human Virol, Baltimore, MD 21201 USA
schillej@dc37a.nci.nih.gov
CYA USA
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (31 JAN 2006) Vol. 103, No. 5, pp. 1516-1521.
ISSN: 0027-8424.
PB NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418 USA.
DT Article; Journal
LA English
REC Reference Count: 69
ED Entered STN: 16 Feb 2006
Last Updated on STN: 16 Feb 2006
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Sexually transmitted human papillomaviruses (HPVs) are the primary cause of cervical cancer. Recent advances in techniques for production of papillomaviral vectors [known as pseudoviruses (PsVs)] have made it possible to perform high-throughput screens for compounds that might block the initial stages of papillomavirus infection. We have used PsVs to screen a variety of compounds that might function as inhibitors of HPV infection, with emphasis on human peptides previously implicated in innate antimicrobial immunity. Little is known about the

possible activity of these peptides against nonenveloped viruses, such as HPVs. Our screen revealed that human alpha-defensins 1-3 [known as human neutrophil peptides (HNPs) 1-3] and human alpha-defensin 5 (HD-5) are potent antagonists of infection by both cutaneous and mucosal papillomavirus types. In contrast, human beta-defensins 1 and 2 displayed little or no anti-HPV activity. HD-5 was particularly active against sexually transmitted HPV types, with 50% inhibitory doses in the high ng/ml range. Microscopic studies of PsV inhibition by the alpha-defensins revealed that they block virion escape from endocytic vesicles but not virion binding or internalization. Consistent with this finding, PsVs remained susceptible to inhibition by alpha-defensins for many hours after initial binding to cells. HNPs 1-3 and HD-5 have been reported to be present in the female genital tract at levels that overlap those that inhibit HPVs in vitro, suggesting that they could present a natural barrier to the sexual transmission of HPV and could serve as the basis of a broad-spectrum topical microbicide.

L12 ANSWER 33 OF 84 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:132705 SCISEARCH
GA The Genuine Article (R) Number: 006BX
TI Innate defences against methicillin-resistant Staphylococcus aureus (MRSA) infection
AU Komatsuzawa H (Reprint); Ouhara K; Yamada S; Fujiwara T; Sayama K; Hashimoto K; Sugai M
CS Hiroshima Univ, Dept Bacteriol, Grad Sch Biomed Sci, Minami Ku, Kasumi 1-2-3, Hiroshima 7348553, Japan (Reprint); Hiroshima Univ, Dept Bacteriol, Grad Sch Biomed Sci, Minami Ku, Hiroshima 7348553, Japan; Hiroshima Univ, Dept Periodontol & Endodontol, Grad Sch Biomed Sci, Minami Ku, Hiroshima 7348553, Japan; Kawasaki Med Univ, Dept Microbiol, Okayama 7010192, Japan; Ehime Univ, Sch Med, Dept Dermatol, Ehime 7910295, Japan
hkomatsu@hiroshima-u.ac.jp
CYA Japan
SO JOURNAL OF PATHOLOGY, (JAN 2006) Vol. 208, No. 2, pp. 249-260.
ISSN: 0022-3417.
PB JOHN WILEY & SONS LTD, THE ATRIUM, SOUTHERN GATE, CHICHESTER PO19 8SQ, W SUSSEX, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 128
ED Entered STN: 9 Feb 2006
Last Updated on STN: 9 Feb 2006
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The innate immune system is the primary defence against bacterial infection. Among the factors involved in innate defence, anti-microbial peptides produced by humans have recently attracted attention due to their relevance to some diseases and also to the development of new chemotherapeutic agents. Staphylococcus aureus is one of the major human pathogens, causing a variety of infections from suppurative disease to food poisoning. Methicillin-resistant S. aureus (MRSA) is a clinical problem and with the recent emergence of a vancomycin-resistant strain, this will pose serious problems in the near future. In investigating the molecular biology of S. aureus infections to develop new chemotherapeutic agents against MRSA infections, knowledge of the interaction of innate anti-microbial peptides with S. aureus is important. In vitro and in vivo experiments demonstrate that exposure of S. aureus to host cells can induce the anti-microbial peptides beta-defensin-2 (hBD2), hBD3, and LL37/CAP18. The induction level of these peptides differs among strains, as does the susceptibility of the strains, with MRSA strains exhibiting lower susceptibility. In summary, the susceptibility of S. aureus strains, including MRSA strains, to components of the innate immune system varies, with the MRSA strains showing more resistance to both innate immune factors and chemotherapeutic agents. Copyright (c) 2006

Pathological Society of Great Britain and Ireland. Published by John Wiley & Sons, Ltd.

L12 ANSWER 34 OF 84 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:973302 SCISEARCH
GA The Genuine Article (R) Number: 967JN
TI Keratinocyte production of cathelicidin provides direct activity against bacterial skin pathogens
AU Braff M H; Zaiou M; Fierer J; Nizet V; Gallo R L (Reprint)
CS 3350 La Jolla Village Dr, Mail Code 151, San Diego, CA 92161 USA (Reprint); Univ Calif San Diego, Dept Med, San Diego, CA 92103 USA; Univ Calif San Diego, Dept Pediat, San Diego, CA 92103 USA; Vet Affairs Med Ctr, San Diego, CA 92161 USA; Univ Henri Poincare, Sch Pharm, Nancy, France
rgallo@vapop.ucsd.edu
CYA USA; France
SO INFECTION AND IMMUNITY, (OCT 2005) Vol. 73, No. 10, pp. 6771-6781. ISSN: 0019-9567.
PB AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA.
DT Article; Journal
LA English
REC Reference Count: 66
ED Entered STN: 6 Oct 2005
Last Updated on STN: 6 Oct 2005
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Immune defense at an interface with the external environment reflects the functions of physical and chemical barriers provided by epithelial and immune cells. Resident epithelial cells, such as keratinocytes, produce numerous peptides with direct antimicrobial activity but also provide a physical barrier against invading pathogens and signal the recruitment of circulating immune cells, such as neutrophils. Antimicrobial peptides such as cathelicidin are produced constitutively by neutropbils and are inducible in keratinocytes in response to infection. The multiplicity of antimicrobial peptides and their cellular sources has resulted in an incomplete understanding of the role of cathelicidin production by epithelial cells in cutaneous immune defense. Therefore, this study sought to evaluate keratinocyte antimicrobial activity and the potential contribution of keratinocyte cathelicidin to host protection against two leading human skin pathogens. Wild-type mice and those with a targeted deletion of the cathelicidin gene, Cnlp, were rendered neutropenic prior to cutaneous infection. Interestingly, Chlp-deficient mice remained more susceptible to group A streptococcus infection than mice with Cnlp intact, suggesting the involvement of epithelial cell-derived cathelicidin in host immune defense. Keratinocytes were then isolated in culture and found to inhibit the growth of Staphylococcus aureus, an effect that was partially dependent on their ability to synthesize and activate cathelicidin. Further, lentivirus-mediated delivery of activated human cathelicidin enhanced keratinocyte antimicrobial activity. Combined, these data illustrate the potential contribution of keratinocyte cathelicidin to the innate immune defense of skin against bacterial pathogens and highlight the need to consider epithelial antimicrobial function in the diagnosis and therapy of skin infection.

L12 ANSWER 35 OF 84 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2004:975665 SCISEARCH
GA The Genuine Article (R) Number: 865PT
TI Host defense peptides in burns
AU Steinstraesser L (Reprint); Oezdogan Y; Wang S C; Steinau H U
CS Ruhr Univ Bochum, BG Univ Hosp Bergmannsheil, Burn Ctr, Dept Plast Surg, Buerkle de la Camp Pl 1, D-44789 Bochum, Germany (Reprint); Ruhr Univ Bochum, BG Univ Hosp Bergmannsheil, Burn Ctr, Dept Plast Surg, D-44789

Bochum, Germany; Univ Michigan, Dept Trauma Burn Surg, Ann Arbor, MI 48109 USA
lars.steinstraesser@ruhr-uni-bochum.de
CYA Germany; USA
SO BURNS, (NOV 2004) Vol. 30, No. 7, pp. 619-627.
ISSN: 0305-4179.
PB ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND.
DT General Review; Journal
LA English
REC Reference Count: 99
ED Entered STN: 2 Dec 2004
Last Updated on STN: 2 Dec 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Overuse of antibiotics and failure to apply basic infection control policies and procedures have contributed to the increasing multi-drug resistance of many nosocomial pathogens. The alarming increase of multi-drug-resistant bacteria (e.g. Pseudomonas aeruginosa, methicilin-resistant Staphylococci, vancomycin-resistant Enterococci) causes infected wounds associated with high mortality and morbidity in burned patients and focuses attention on the need for better treatment and prevention of wound infections.

The review points out and discusses some emerging alternatives to antibiotics used in clinical practice, with special emphasis on the role of the innate immune response and potential application of human host defense peptides in thermal injury.

L12 ANSWER 36 OF 84 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2001:842928 SCISEARCH
GA The Genuine Article (R) Number: 483ZC
TI Susceptibilities of oral bacteria and yeast to mammalian cathelicidins
AU Guthmiller J M (Reprint); Vargas K G; Srikantha R; Schomberg L L; Weistroffer P L; McCray P B; Tack B F
CS Univ Iowa, Coll Dent, Dept Periodont, Iowa City, IA 52242 USA (Reprint); Univ Iowa, Coll Dent, Dept Pediat Dent, Iowa City, IA 52242 USA; Univ Iowa, Coll Dent, Dows Inst Dent Res, Iowa City, IA 52242 USA; Univ Iowa, Coll Med, Dept Pediat, Iowa City, IA 52242 USA; Univ Iowa, Coll Med, Dept Microbiol, Iowa City, IA 52242 USA
CYA USA
SO ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, (NOV 2001) Vol. 45, No. 11, pp. 3216-3219.
ISSN: 0066-4804.
PB AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA.
DT Article; Journal
LA English
REC Reference Count: 25
ED Entered STN: 2 Nov 2001
Last Updated on STN: 2 Nov 2001
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The effects of cathelicidins against oral bacteria and clinically important oral yeasts are not known. We tested the susceptibilities of Actinobacillus actinomycetemcomitans, Fusobacterium nucleatum, Porphyromonas gingivalis, Streptococcus sanguis, Candida krusei, Candida tropicalis and Candida albicans to the following cathelicidins: FALL39, SMAP29, and CAP18. SMAP29 and CAP18 were antimicrobial, whereas FALL39 did not exhibit antimicrobial activity. Future studies are needed to determine the potential use of these antimicrobial peptides in prevention and treatment of oral infections.

L12 ANSWER 37 OF 84 USPATFULL on STN

AN 2007:75557 USPATFULL
TI Human cathelicidin antimicrobial peptides
IN Gallo, Richard, San Diego, CA, UNITED STATES
Murakami, Masamoto, Asahikawa, JAPAN
PA The Regents of the University of California, Oakland, CA, UNITED STATES, 94607-5200 (U.S. corporation)
PI US 2007065908 A1 20070322
AI US 2004-575537 A1 20041020 (10)
WO 2004-US34911 20041020
20060829 PCT 371 date
PRAI US 2003-512953P 20031021 (60)
DT Utility
FS APPLICATION
LREP BUCHANAN, INGERSOLL & ROONEY LLP, P.O. BOX 1404, ALEXANDRIA, VA, 22313-1404, US
CLMN Number of Claims: 68
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 2326
AB Provided are peptide and peptide consensus sequences, which inhibit bacterial growth and/or viral growth and mimic the activity of LL-37, CRAMP, and/or FALL-39. The peptides are useful as antimicrobials, ant-inflammatories and antiviral agents.

L12 ANSWER 38 OF 84 USPATFULL on STN
AN 2007:68445 USPATFULL
TI Method for determining the susceptibility of a subject to infection
IN Boman, Hans G., Stockholm, SWEDEN
Andersson, Mats, Stockholm, SWEDEN
Putsep, Katrin, Stockholm, SWEDEN
Carlsson, Goran, Stockholm, SWEDEN
PI US 2007059691 A1 20070315
AI US 2003-530606 A1 20031010 (10)
WO 2003-EP11240 20031010
20060221 PCT 371 date
PRAI GB 2002-23655 20021010
DT Utility
FS APPLICATION
LREP HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US
CLMN Number of Claims: 32
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 1179
AB A method for determining the susceptibility of a subject to infection, which method comprises: (i) providing a sample from said subject; (ii) detecting any LL-37 present in said sample; (iii) optionally comparing the level of LL-37 in said sample to a control sample; and (iv) determining the susceptibility of said subject to infection, wherein no LL-37 or a low level of LL-37 indicates that said subject is susceptible to infection.

L12 ANSWER 39 OF 84 USPATFULL on STN
AN 2007:56572 USPATFULL
TI Novel method of treatment of inflammatory skin conditions
IN Chandler, Stephen Rupert, Abingdon, UNITED KINGDOM
Layton, Guy Timothy, Abingdon, UNITED KINGDOM
PI US 2007049518 A1 20070301
AI US 2006-515373 A1 20060831 (11)
PRAI GB 2005-17685 20050831
GB 2006-13954 20060714

DT Utility
FS APPLICATION
LREP KLAUBER & JACKSON, 411 HACKENSACK AVENUE, HACKENSACK, NJ, 07601, US
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 2 Drawing Page(s)
LN.CNT 1629
AB There is provided, inter alia, a method for the treatment or prevention of an inflammatory skin condition which is characterised by colonisation with Staphylococcus aureus, comprising the topical administration of an aureolysin inhibitor.

L12 ANSWER 40 OF 84 USPATFULL on STN
AN 2007:43045 USPATFULL
TI Human cathelicidin antimicrobial peptides
IN Gallo, Richard L., San Diego, CA, UNITED STATES
Murakami, Masamoto, Asahikawa, JAPAN
Leung, Donald Y.M., Denver, CO, UNITED STATES
PA The Regents of the University of California, Oakland, CA, UNITED STATES (U.S. corporation)
PI US 2007037744 A1 20070215
AI US 2004-575552 A1 20041020 (10)
WO 2004-US34948 20041020
20060829 PCT 371 date
PRAI US 2003-512953P 20031021 (60)
DT Utility
FS APPLICATION
LREP BUCHANAN, INGERSOLL & ROONEY LLP, P.O. BOX 1404, ALEXANDRIA, VA, 22313-1404, US
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 2028
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Provided are peptide and peptide consensus sequences, which inhibit bacterial growth and/or viral growth and mimic the activity of LL-37, CRAMP, and/or FALL-39. The peptides are useful as antimicrobials, anti-inflammatories and anti-viral agents.

L12 ANSWER 41 OF 84 USPATFULL on STN
AN 2007:36425 USPATFULL
TI Anti-pathogen treatments
IN Rider, Todd H., Littleton, MA, UNITED STATES
PI US 2007031965 A1 20070208
AI US 2006-503416 A1 20060811 (11)
RLI Division of Ser. No. US 2003-361208, filed on 7 Feb 2003, GRANTED, Pat. No. US 7125839
PRAI US 2002-355359P 20020207 (60)
US 2002-355022P 20020207 (60)
US 2002-432386P 20021210 (60)
DT Utility
FS APPLICATION
LREP HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 86 Drawing Page(s)
LN.CNT 11787
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Chimeric molecules that contain at least one pathogen-detection domain and at least one effector domain, and their methods of use in preventing or treating a pathogen infection in a cell or organism are described. The pathogen-detection domain and effector domain of the chimeric molecules are domains not typically found in nature to be

associated together. Agents are also described herein having at least one pathogen-interacting molecular structure and at least one effector-mediating molecular structure, the agent being one that is non-naturally-occurring in a cell. The methods of prevention and treatment described herein are effective for a broad spectrum of pathogens and exhibit little or no toxic side-effects. Assays for the detection of a pathogen, pathogen component, or product produced or induced by a pathogen, are also provided.

L12 ANSWER 42 OF 84 USPATFULL on STN
AN 2007:30178 USPATFULL
TI Polynucleotide encoding a novel human G-protein coupled receptor, HBPRBMY39
IN Ramanathan, Chandra S., Ringoes, NJ, UNITED STATES
Gopal, Shuba, Rochester, NY, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
PA Bristol-Myers Squibb Company (U.S. corporation)
PI US 2007026448 A1 20070201
AI US 2006-515921 A1 20060905 (11)
RLI Division of Ser. No. US 2002-237813, filed on 6 Sep 2002, PENDING
PRAI US 2001-317793P 20010907 (60)
US 2001-333658P 20011127 (60)
DT Utility
FS APPLICATION
LREP LOUIS J. WILLE, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000, US
CLMN Number of Claims: 11
ECL Exemplary Claim: 1-19
DRWN 11 Drawing Page(s)
LN.CNT 14690
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding HGPRBMY39 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel HGPRBMY39 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

L12 ANSWER 43 OF 84 USPATFULL on STN
AN 2006:340770 USPATFULL
TI Anti-viral activity of cathelicidin peptides
IN Gallo, Richard L., San Diego, CA, UNITED STATES
Leung, Donald Y. M., Denver, CO, UNITED STATES
Jones, James F., Decatur, GA, UNITED STATES
PI US 2006292551 A1 20061228
AI US 2004-546739 A1 20040305 (10)
WO 2004-US6952 20040305
20060731 PCT 371 date
PRAI US 2003-452906P 20030306 (60)
DT Utility
FS APPLICATION
LREP BUCHANAN, INGERSOLL & ROONEY LLP, P.O. BOX 1404, ALEXANDRIA, VA, 22313-1404, US
CLMN Number of Claims: 50
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 1609
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The disclosure provides methods and compositions useful in the treatment of dermatitis and viral infections. The compositions comprise

cationic peptides of the cathelicidin family including LL-37, related homologues, and variants thereof.

L12 ANSWER 44 OF 84 USPATFULL on STN
AN 2006:282088 USPATFULL
TI Assay
IN Akesson, Per, Lund, SWEDEN
Bjorck, Lars, Lund, SWEDEN
Sjoholm, Anders, Lund, SWEDEN
PI US 2006241031 A1 20061026
AI US 2004-545904 A1 20040219 (10)
WO 2004-EP1581 20040219
20051122 PCT 371 date
PRAI GB 2003-3809 20030219
DT Utility
FS APPLICATION
LREP FOLEY AND LARDNER LLP, SUITE 500, 3000 K STREET NW, WASHINGTON, DC, 20007, US
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 808
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An assay method for an anti-bacterial agent comprising: (a) providing as a first component protein SIC; (b) providing as a second component an antibacterial peptide; (c) contacting the first component with a test substance in the presence of the second component; and (d) determining the interaction or activity of the first component with the second component to determine thereby whether a test substance is an effective anti-bacterial agent.

L12 ANSWER 45 OF 84 USPATFULL on STN
AN 2006:267697 USPATFULL
TI Novel microorganism Pediococcus pentosaceus EROM101, having immune enhancement, anticancer and antimicrobial activities
IN Choi, Chang Won, Gyeonggi-do, KOREA, REPUBLIC OF
Park, Mi Hyoun, Seoul, KOREA, REPUBLIC OF
Hwang, Sang Ho, Seoul, KOREA, REPUBLIC OF
Woo, Suk Gyu, Gyeonggi-do, KOREA, REPUBLIC OF
Song, Mi Kyung, Seoul, KOREA, REPUBLIC OF
Im, Jong Jun, Seoul, KOREA, REPUBLIC OF
Hong, Sung Gil, Seoul, KOREA, REPUBLIC OF
Kim, Joong Hark, Seoul, KOREA, REPUBLIC OF
Jang, Jung Soon, Seoul, KOREA, REPUBLIC OF
Kim, Hwa Young, Seoul, KOREA, REPUBLIC OF
PA EROMLIFE CO., LTD., Seoul, KOREA, REPUBLIC OF, 135-010 (non-U.S. corporation)
PI US 2006228379 A1 20061012
AI US 2006-450452 A1 20060612 (11)
RLI Continuation of Ser. No. US 2003-732340, filed on 11 Dec 2003, PENDING
DT Utility
FS APPLICATION
LREP BUCHANAN, INGERSOLL & ROONEY PC, POST OFFICE BOX 1404, ALEXANDRIA, VA, 22313-1404, US
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 933
AB The present invention relates to a novel Pediococcus genus microorganism and more particularly, Pediococcus pentosaceus EROM101 (KCCM-10517) originated from human intestines having immune enhancement, anticancer and antiviral activities and a use thereof. Due to its excellent immune enhancement, anticancer and antimicrobial activities by activating macrophages/spleen cells and inducing gut immunity, the Pediococcus

pentosaceus EROM101 of the present invention can be effectively used for the production of various products such as immune enhancement agent, anticancer agent, antimicrobial agent, food additive, intestinal function-controlling agent, live bacterial agent, feed additive and other fermented products.

L12 ANSWER 46 OF 84 USPATFULL on STN
AN 2006:261122 USPATFULL
TI Methods and compositions for selecting cells with increased potency
IN Centanni, John M., Madison, WI, UNITED STATES
Allen-Hoffmann, Lynn, Madison, WI, UNITED STATES
PI US 2006222635 A1 20061005
AI US 2005-297916 A1 20051209 (11)
RLI Division of Ser. No. US 2004-909119, filed on 30 Jul 2004, PENDING
PRAI US 2003-491869P 20030801 (60)
US 2003-493664P 20030808 (60)
DT Utility
FS APPLICATION
LREP MEDLEN & CARROLL, LLP, Suite 350, 101 Howard Street, San Francisco, CA, 94105, US
CLMN Number of Claims: 14
ECL Exemplary Claim: 1-80
DRWN 22 Drawing Page(s)
LN.CNT 4742
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to compositions for wound closure. More specifically, the present invention provides human skin equivalents engineered to express exogenous polypeptides (e.g., antimicrobial polypeptides and keratinocyte growth factor 2) and compositions and methods for making human skin equivalents engineered to express exogenous polypeptides. In addition, the present invention provides methods for treatment of wounds with human skin equivalents engineered to express exogenous polypeptides.

L12 ANSWER 47 OF 84 USPATFULL on STN
AN 2006:241198 USPATFULL
TI Methods for increasing cell and tissue viability
IN Sanders, Mitchell C., West Boylston, MA, UNITED STATES
Ellis-Busby, Diane L., Lancaster, MA, UNITED STATES
Sebastian, Shite, Somerville, MA, UNITED STATES
PI US 2006205646 A1 20060914
AI US 2005-271158 A1 20051111 (11)
RLI Continuation of Ser. No. WO 2004-US14920, filed on 12 May 2004, PENDING
PRAI US 2004-537814P 20040121 (60)
US 2003-469869P 20030512 (60)
DT Utility
FS APPLICATION
LREP HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133, US
CLMN Number of Claims: 47
ECL Exemplary Claim: 1
DRWN 33 Drawing Page(s)
LN.CNT 3440
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention features methods of increasing cell or tissue viability by administering to the cell or tissue a protective protein. The invention also features methods of treating a condition characterized by cell or tissue damage in a subject by administering to the subject a protective protein. Also included are chimeric proteins as well as methods of inhibiting proteolysis of a cationic antimicrobial peptide in a cell or tissue including contacting the cell or tissue with a protective protein, chimeric protein that includes the protective protein, or a biologically active fragment, variant, or derivative thereof.

L12 ANSWER 48 OF 84 USPATFULL on STN
AN 2006:222415 USPATFULL
TI Use of xylitol to reduce ionic strength and activate endogenous antimicrobials for prevention and treatment of infections
IN Welsh, Michael J., Riverside, IA, UNITED STATES
Zabner, Joseph, Iowa City, IA, UNITED STATES
PA UNIVERSITY OF IOWA RESEARCH FOUNDATION, Iowa City, IA, UNITED STATES (U.S. corporation)
PI US 2006189701 A1 20060824
AI US 2006-405345 A1 20060417 (11)
RLI Continuation of Ser. No. US 2004-766506, filed on 28 Jan 2004, PENDING
Continuation of Ser. No. US 2001-861841, filed on 21 May 2001, GRANTED, Pat. No. US 6716819
PRAI US 2000-205948P 20000519 (60)
DT Utility
FS APPLICATION
LREP MCKEE, VOORHEES & SEASE, P.L.C., 801 GRAND AVENUE, SUITE 3200, DES MOINES, IA, 50309-2721, US
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 6 Drawing Page(s)
LN.CNT 1165
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for killing infectious microbial cells by exposing the microbial cells to endogenous antimicrobial compounds. Activation of the antimicrobials is achieved by addition of low permeability, non-ionic osmolytes to lower ionic strength in body fluids where the antimicrobials have been previously suppressed by alteration of ionic transport (increase in salt concentration). The method can be used to treat cystic fibrosis. Cystic fibrosis causes elevated salt concentrations in the airway surface liquid (ASL) occur due to the impaired chloride transport across the epithelia. Xylitol has been found to be an effective low permeability, non-ionic osmolyte for use in the present invention.

L12 ANSWER 49 OF 84 USPATFULL on STN
AN 2006:203054 USPATFULL
TI ANTIMICROBIAL AGENT
IN KIM, Yeon Sook, Department of Oral Pathology, College of Dentistry, Kangnung National University, Chibyon-dong, Gangneung, Gangwon-do, KOREA, REPUBLIC OF 210-702
LEE, Suk Keun, Department of Oral Pathology, College of Dentistry, Kangnung National University, Chibyon-dong, Gangneung, Gangwon-do, KOREA, REPUBLIC OF 210-702
CHUNG, Soo Il, 6839 Old Stage Rd., Rockville, MD, UNITED STATES 20852-4359
PA CORAM BIOSCIENCE, INC., Rockville, MD, UNITED STATES (U.S. corporation)
PI US 2006172940 A1 20060803
AI US 2006-307316 A1 20060131 (11)
PRAI US 2005-648815P 20050131 (60)
DT Utility
FS APPLICATION
LREP JHK LAW, P.O. BOX 1078, LA CANADA, CA, 91012-1078, US
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 2273
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present application discloses a therapeutic antimicrobial composition comprising mucocidin antimicrobial peptides or analogue or fragments thereof having antimicrobial activity.

L12 ANSWER 50 OF 84 USPATFULL on STN

AN 2006:203045 USPATFULL
TI Heparin-binding peptides and uses thereof
IN San Antonio, James D., Media, PA, UNITED STATES
Schick, Barbara P., Merion Station, PA, UNITED STATES
Verrecchio, Angela, Philadelphia, PA, UNITED STATES
PA Thomas Jefferson University, Philadelpia, PA, UNITED STATES, 19107 (U.S. corporation)
PI US 2006172931 A1 20060803
AI US 2004-551005 A1 20040329 (10)
WO 2004-US9668 20040329
20060112 PCT 371 date
PRAI US 2003-458241P 20030328 (60)
DT Utility
FS APPLICATION
LREP DRINKER BIDDLE & REATH, ATTN: INTELLECTUAL PROPERTY GROUP, ONE LOGAN SQUARE, 18TH AND CHERRY STREETS, PHILADELPHIA, PA, 19103-6996, US
CLMN Number of Claims: 65
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 2860
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Heparin-binding peptides are provided of the formula R.sub.1(X.sub.1B.sub.1B.sub.2X.sub.2B.sub.3X.sub.3Y.sub.1R.sub.2).sub.nR.sub.3, R.sub.1(X.sub.1B.sub.1B.sub.2B.sub.3X.sub.2X.sub.3B.sub.4X.sub.4Y.sub.1R.sub.2).sub.nR.sub.3, and C(X.sub.1B.sub.1B.sub.2B.sub.3X.sub.2X.sub.3B.sub.4X.sub.4).sub.nC; wherein X.sub.1, X.sub.2, X.sub.3, and X.sub.4 are independently selected from the group consisting of hydropathic amino acids; B.sub.1, B.sub.2, B.sub.3, and B.sub.4 are independently selected from the group consisting of basic amino acids; C is cysteine; Y.sub.1 is zero or one to ten amino acid residues, wherein at least one amino acid residue is proline; n is an integer from one to ten; and R.sub.1, R.sub.2, and R.sub.3 are independently selected segments containing from zero to twenty amino acid residues, provided, at least one of the segments R.sub.1, R.sub.2, and R.sub.3 comprises at least one hydrophobic amino acid residue. The peptide C(X.sub.1B.sub.1B.sub.2B.sub.3X.sub.2X.sub.3B.sub.4X.sub.4).sub.nC is optionally cyclized via a disulfide bond formed between cysteine residues. The peptides are administered to reduce plasma LMWH and heparin levels and to reduce the anticoagulant effects of heparin and LMWH. The peptides are also administered to inhibit microbial growth and to inhibit mast cell serine proteases involved in various diseases and disorders. The peptides are also administered as carriers to deliver active agents.

L12 ANSWER 51 OF 84 USPATFULL on STN
AN 2006:173997 USPATFULL
TI Biocides
IN Homan, Jane, Hillpoint, WI, UNITED STATES
Imboden, Michael, Madison, WI, UNITED STATES
Riggs, Michael, Tuscon, AZ, UNITED STATES
Carryn, Stephane, Brussels, BELGIUM
Schaefer, Deborah A., Tuscon, AZ, UNITED STATES
PA ioGenetics, Madison, WI, UNITED STATES (U.S. corporation)
University of Arizona, Tucson, AZ, UNITED STATES (U.S. corporation)
PI US 2006147442 A1 20060706
AI US 2005-254500 A1 20051020 (11)
RLI Continuation-in-part of Ser. No. US 2004-844837, filed on 13 May 2004, PENDING
PRAI US 2003-470841P 20030515 (60)
US 2004-620642P 20041020 (60)
DT Utility
FS APPLICATION
LREP Medlen & Carroll, LLP, Suite 350, 101 Howard Street, San Francisco, CA, 94105, US

CLMN Number of Claims: 30
ECL Exemplary Claim: 1
DRWN 23 Drawing Page(s)
LN.CNT 6715
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to the use of biocide (e.g., bactericidal enzyme) to target pathogens. In particular, the present invention provides biocides for use in health care (e.g., human and veterinary), agriculture (e.g., animal and plant production), and food processing (e.g., water purification).

L12 ANSWER 52 OF 84 USPATFULL on STN
AN 2006:170000 USPATFULL
TI Alpha helical peptides with broad spectrum antimicrobial activity that are insensitive to salt
IN Tack, Brian F., Iowa City, IA, UNITED STATES
McCray, Jr., Paul B., Iowa City, IA, UNITED STATES
Welsh, Michael, Riverside, IA, UNITED STATES
Travis, Sue M., Iowa City, IA, UNITED STATES
Lehrer, Robert, Santa Monica, CA, UNITED STATES
PA The University of Iowa Research Foundation, Iowa City, IA, UNITED STATES (U.S. corporation)
The Regents of the University of California, Oakland, CA, UNITED STATES (U.S. corporation)
PI US 7071293 B1 20060704
AI US 2000-642744 20000818 (9)
PRAI US 1999-149886P 19990818 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Minnifield, N. M.
LREP Fulbright & Jaworski
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 46 Drawing Figure(s); 21 Drawing Page(s)
LN.CNT 3520
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to the use of antimicrobial peptides in the inhibition of microbial growth and proliferation. Novel antimicrobial truncated peptides are disclosed which are based upon SMAP 29 and RCAP 18, but which contain a lesser number of amino acid residues yet still retain bactericidal activity. In addition, synthetic peptides based upon the SMAP 29 protein are disclosed which have fewer amino acid residues and include substitutions yet retain substantial activity. The invention also relates to a method of inhibiting microbial growth by administering an effective amount of a peptide in accordance with the invention, or by combining the peptides with other antimicrobial agents or antibiotics.

L12 ANSWER 53 OF 84 USPATFULL on STN
AN 2006:159100 USPATFULL
TI Method for screening for an antimicrobial polypeptide
IN Segura, Dorotea Raventos, Humlebaek, DENMARK
Hansen, Anja Lykke, Copenhagen V, DENMARK
Mygind, Per Holse, Soborg, DENMARK
Hogenhaug, Hans-Henrik Kristensen, Holte, DENMARK
Ellingsgaard, Ida, Lyngby, DENMARK
PA Novozymes A/S, Bagsvaerd, DENMARK (non-U.S. corporation)
PI US 2006134630 A1 20060622
AI US 2003-682104 A1 20031009 (10)
PRAI DK 2002-1518 20021009
DK 2002-1854 20021202
US 2002-419050P 20021016 (60)
DT Utility
FS APPLICATION

LREP NOVOZYMES NORTH AMERICA, INC., 500 FIFTH AVENUE, SUITE 1600, NEW YORK, NY, 10110, US
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2290
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a method for screening polynucleotide sequences encoding antimicrobial polypeptides and methods for testing the antimicrobial activity of an antimicrobial polypeptide.

L12 ANSWER 54 OF 84 USPATFULL on STN
AN 2006:98972 USPATFULL
TI Polynucleotide encoding a novel human G-protein coupled receptor, HGPRBMY39
IN Ramanathan, Chandra S., Wallingford, CT, UNITED STATES
Gopal, Shuba, New York, NY, UNITED STATES
Mintier, Gabriel, Hightstown, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
PI US 2006084140 A1 20060420
AI US 2002-237813 A1 20020906 (10)
PRAI US 2001-317793P 20010907 (60)
US 2001-333658P 20011127 (60)
DT Utility
FS APPLICATION
LREP LOUIS J. WILLE, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000, US
CLMN Number of Claims: 17
ECL Exemplary Claim: 1-19
DRWN 11 Drawing Page(s)
LN.CNT 14731
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding HGPRBMY39 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel HGPRBMY39 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

L12 ANSWER 55 OF 84 USPATFULL on STN
AN 2006:87036 USPATFULL
TI Tryptophan as a functional replacement for adp-ribose-arginine in recombinant proteins
IN Moss, Joel, Bethesda, MD, UNITED STATES
Stevens, Linda, Gaithersburg, MD, UNITED STATES
Bourgeois, Christelle, Bethesda, MD, UNITED STATES
Bortell, Rita, Shirley, MA, UNITED STATES
PI US 2006074037 A1 20060406
AI US 2003-517565 A1 20030627 (10)
WO 2003-US20498 20030627
20041207 PCT 371 date
PRAI US 2002-393033P 20020628 (60)
DT Utility
FS APPLICATION
LREP KLARQUIST SPARKMAN, LLP, 121 S.W. SALMON STREET, SUITE #1600, PORTLAND, OR, 97204-2988, US
CLMN Number of Claims: 35
ECL Exemplary Claim: 1
DRWN 6 Drawing Page(s)
LN.CNT 2295

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method is disclosed for producing a polypeptide with a modified activity or stability, by replacing an arginine residue capable of being ADP-ribosylated with a tryptophan or a phenylalanine. In one embodiment, compositions are provided that include polypeptides, such as alpha defensin, with arginine-to-tryptophan or arginine-to-phenylalanine substitutions, where the arginine residue is capable of being ADP-ribosylated. In another embodiment, methods are disclosed for modifying an immune response in a subject.

L12 ANSWER 56 OF 84 USPATFULL on STN
AN 2006:81027 USPATFULL
TI D-isomers of antimicrobial peptide
IN Bobek, Libuse A., Williamsville, NY, UNITED STATES
PI US 2006069022 A1 20060330
AI US 2005-213245 A1 20050826 (11)
PRAI US 2004-606312P 20040901 (60)
DT Utility
FS APPLICATION
LREP HODGSON RUSS LLP, ONE M & T PLAZA, SUITE 2000, BUFFALO, NY, 14203-2391, US
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 1145
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides D-isomers of MUC7-12-mer peptide of human saliva MUC7. The isomers have antimicrobial activity comparable to that of the L-isomers and are resistant to proteolysis. These peptides can be used as antifungal and antimicrobial agents.

L12 ANSWER 57 OF 84 USPATFULL on STN
AN 2006:74177 USPATFULL
TI Enzymatic activities in chemokine-mediated inflammation
IN Berahovich, Robert D., Berkeley, CA, UNITED STATES
Miao, Zhenhua, San Jose, CA, UNITED STATES
Premack, Brett, San Francisco, CA, UNITED STATES
Schall, Thomas J., Palo Alto, CA, UNITED STATES
PI US 2006063223 A1 20060323
AI US 2005-198935 A1 20050804 (11)
PRAI US 2004-598959P 20040804 (60)
DT Utility
FS APPLICATION
LREP BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610, US
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN 25 Drawing Page(s)
LN.CNT 4685
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Truncated chemokines lacking an N-terminal region that activate CCR1 and/or FPRL1 and compositions containing the truncated chemokines are provided. Methods of identifying agents that modulate CCR1 and/or FPRL1 activity either by modulating the production of the truncated chemokines or the ability of the truncated chemokines to activate CCR1 and/or FPRL1 are also disclosed. Methods using the truncated chemokines to inhibit or activate CCR1 and/or FPRL1 mediated biological activities are also disclosed.

L12 ANSWER 58 OF 84 USPATFULL on STN
AN 2006:68045 USPATFULL
TI Chemically modified oligonucleotides
IN Manoharan, Muthiah, Weston, MA, UNITED STATES
Kesavan, Venkitasamy, Woburn, MA, UNITED STATES
Rajeev, Kallanthottathil G., Cambridge, MA, UNITED STATES

PI US 2006058266 A1 20060316
AI US 2005-200703 A1 20050810 (11)
PRAI US 2004-600703P 20040810 (60)
DT Utility
FS APPLICATION
LREP FISH & RICHARDSON PC, P.O. BOX 1022, MINNEAPOLIS, MN, 55440-1022, US
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 25 Drawing Page(s)
LN.CNT 6838
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates composition and methods for making and using chemically modified oligonucleotides agents for inhibiting gene expression.

L12 ANSWER 59 OF 84 USPATFULL on STN
AN 2006:67435 USPATFULL
TI Methods for determining susceptibility to dental caries
IN Dale-Crunk, Beverly A., Ellensburg, WA, UNITED STATES
Kimball, Janet R., Seattle, WA, UNITED STATES
Tao, Renchuan, Guangxi, CHINA
PI US 2006057654 A1 20060316
AI US 2005-214474 A1 20050829 (11)
PRAI US 2004-610579P 20040916 (60)
DT Utility
FS APPLICATION
LREP CHRISTENSEN, O'CONNOR, JOHNSON, KINDNESS, PLLC, 1420 FIFTH AVENUE, SUITE 2800, SEATTLE, WA, 98101-2347, US
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 597
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods for determining whether a human being is susceptible to dental caries. The methods each include the steps of measuring the amount of α-defensins HNP 1, HNP 2 and HNP 3 in saliva obtained from a human being, and determining whether a reduced amount of the α-defensins HNP 1, HNP 2 and HNP 3 is present in the saliva, thereby determining whether the human being is susceptible to dental caries.

L12 ANSWER 60 OF 84 USPATFULL on STN
AN 2005:293520 USPATFULL
TI IRNA agents with biocleavable tethers
IN Manoharan, Muthiah, Weston, MA, UNITED STATES
Rajeev, Kallanthottathil G., Cambridge, MA, UNITED STATES
PI US 2005256069 A1 20051117
AI US 2004-985426 A1 20041109 (10)
RLI Continuation-in-part of Ser. No. US 2004-916185, filed on 10 Aug 2004, PENDING Continuation-in-part of Ser. No. WO 2004-US11829, filed on 16 Apr 2004, PENDING
PRAI WO 2004-US7070 20040308
WO 2004-US10586 20040405
WO 2004-US11255 20040409
WO 2004-US11822 20040416
US 2003-493986P 20030808 (60)
US 2003-494597P 20030811 (60)
US 2003-506341P 20030926 (60)
US 2003-518453P 20031107 (60)
US 2003-463772P 20030417 (60)
US 2003-465802P 20030425 (60)
US 2003-469612P 20030509 (60)
US 2003-510246P 20031009 (60)
US 2003-510318P 20031010 (60)

US 2003-503414P 20030915 (60)
US 2003-465665P 20030425 (60)
DT Utility
FS APPLICATION
LREP FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
CLMN Number of Claims: 101
ECL Exemplary Claim: 1
DRWN 15 Drawing Page(s)
LN.CNT 6764
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to iRNA agents, which preferably include a monomer in which the ribose moiety has been replaced by a moiety other than ribose that further includes a tether having one or more linking groups, in which at least one of the linking groups is a cleavable linking group. The tether in turn can be connected to a selected moiety, e.g., a ligand, e.g., a targeting or delivery moiety, or a moiety which alters a physical property. The cleavable linking group is one which is sufficiently stable outside the cell such that it allows targeting of a therapeutically beneficial amount of an iRNA agent (e.g., a single stranded or double stranded iRNA agent), coupled by way of the cleavable linking group to a targeting agent--to targets cells, but which upon entry into a target cell is cleaved to release the iRNA agent from the targeting agent. The inclusion of such a monomer can allow for modulation of a property of the iRNA agent into which it is incorporated, e.g., by using the non-ribose moiety as a point to which a ligand or other entity, e.g., a lipophilic moiety. e.g., cholesterol, is is directly, or indirectly, tethered. The invention also relates to methods of making and using such modified iRNA agents.

L12 ANSWER 61 OF 84 USPATFULL on STN
AN 2005:268684 USPATFULL
TI Compositions and methods of use of W-peptides
IN Premack, Brett, San Francisco, CA, UNITED STATES
Schall, Thomas, Palo Alto, CA, UNITED STATES
PI US 2005234004 A1 20051020
AI US 2005-43020 A1 20050125 (11)
PRAI US 2004-539665P 20040126 (60)
DT Utility
FS APPLICATION
LREP BRINKS HOFER GILSON & LIONE, P.O. BOX 10395, CHICAGO, IL, 60610, US
CLMN Number of Claims: 54
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1655
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to compositions and methods to modulating immune responses, such as those elicited by vaccination with W peptides. The compositions and methods are useful for, among other things, vaccine formulation for therapeutic and prophylactic vaccination (immunization) and for production of useful antibodies (e.g., monoclonal antibodies, for therapeutic or diagnostic use).

L12 ANSWER 62 OF 84 USPATFULL on STN
AN 2005:233095 USPATFULL
TI Family of cystatin-related chemoattractant proteins
IN Zabel, Brian A., Mountain View, CA, UNITED STATES
Butcher, Eugene C., Portola Valley, CA, UNITED STATES
PA The Board of Trustees of the Leland Stanford Junior University (U.S. corporation)
PI US 2005202029 A1 20050915
AI US 2004-958527 A1 20041004 (10)
PRAI US 2003-508360P 20031003 (60)
DT Utility
FS APPLICATION

LREP BOZICEVIC, FIELD & FRANCIS LLP, 1900 UNIVERSITY AVENUE, SUITE 200, EAST PALO ALTO, CA, 94303, US
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 6 Drawing Page(s)
LN.CNT 2317
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided to modulate the trafficking of leukocytes through interactions with one or more of a class of chemoattractant proteins having a cystatin-like structure. Exemplary of proteins in this class is chemerin, which interacts with the receptor CMKLR1. The chemoattractant polypeptide, or agonists of the chemoattractant receptor, act to concentrate responding leukocytes at a site of interest. Agonists and antagonists of the chemoattractant modulate immune responsiveness.

L12 ANSWER 63 OF 84 USPATFULL on STN
AN 2005:221484 USPATFULL
TI Methods and compositions related to plunc polypeptides
IN McCray, Paul B. JR., Iowa City, IA, UNITED STATES
Weiss, Jerry, Coralville, IA, UNITED STATES
Jia, Hong Peng, Iowa City, IA, UNITED STATES
Schutte, Brian, Iowa City, IA, UNITED STATES
Bartlett, Jennifer, Coralville, IA, UNITED STATES
PI US 2005192221 A1 20050901
AI US 2004-3126 A1 20041203 (11)
PRAI US 2003-526882P 20031204 (60)
DT Utility
FS APPLICATION
LREP FULBRIGHT & JAWORSKI L.L.P., 600 CONGRESS AVE., SUITE 2400, AUSTIN, TX, 78701, US
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 16 Drawing Page(s)
LN.CNT 3028
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention concerns compositions and methods related to the use of peptides and polypeptides corresponding to all or part of the SPLUNC1 and LPLUNC1 proteins. Particular aspects of the invnetion include the use of SPLUNC1 and LPLUNC1 peptides and polypeptides as antimicrobials, anti-inflammatory, and immune modulatory agents.

L12 ANSWER 64 OF 84 USPATFULL on STN
AN 2005:214917 USPATFULL
TI Method of treating neurodegenerative disease
IN Bumcrot, David, Belmont, MA, UNITED STATES
Farrer, Matthew J., Jacksonville Beach, FL, UNITED STATES
Maraganore, Demetrius M., Rochester, MN, UNITED STATES
Vornlocher, Hans-Peter, Bayreuth, GERMANY, FEDERAL REPUBLIC OF
PA Alnylam Pharmaceuticals (U.S. corporation)
Mayo Foundation for Medical Education and Research (U.S. corporation)
PI US 2005186591 A1 20050825
AI US 2004-991286 A1 20041117 (10)
RLI Continuation-in-part of Ser. No. WO 2004-US18271, filed on 9 Jun 2004, PENDING
PRAI US 2003-476947P 20030609 (60)
DT Utility
FS APPLICATION
LREP FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
CLMN Number of Claims: 71
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 6422
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Aspects featured in the invention relate to compositions and methods for

inhibiting alpha-synuclein (SNCA) gene expression, such as for the treatment of neurodegenerative disorders. An anti-SNCA agent featured herein that targets the SNCA gene can have been modified to alter distribution in favor of neural cells.

L12 ANSWER 65 OF 84 USPATFULL on STN
AN 2005:177257 USPATFULL
TI iRNA conjugates
IN Manoharan, Muthiah, Weston, MA, UNITED STATES
PI US 2005153337 A1 20050714
AI US 2004-4379 A1 20041203 (11)
RLI Continuation of Ser. No. WO 2004-US10586, filed on 5 Apr 2004, PENDING
PRAI WO 2004-US7070 20040308
US 2003-460783P 20030403 (60)
US 2003-462894P 20030414 (60)
US 2003-463772P 20030417 (60)
US 2003-465665P 20030425 (60)
US 2003-465802P 20030425 (60)
US 2003-469612P 20030509 (60)
US 2003-493986P 20030808 (60)
US 2003-494597P 20030811 (60)
US 2003-503414P 20030915 (60)
US 2003-506341P 20030926 (60)
US 2003-510246P 20031009 (60)
US 2003-510318P 20031010 (60)
US 2003-518453P 20031107 (60)
DT Utility
FS APPLICATION
LREP FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN 16 Drawing Page(s)
LN.CNT 8629
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Therapeutic sRNA agents and methods of making and using are enclosed.

L12 ANSWER 66 OF 84 USPATFULL on STN
AN 2005:151376 USPATFULL
TI Novel microorganism Pediococcus pentosaceus EROM101, having immune enhancement, anticancer and antimicrobial activities
IN Choi, Chang Won, Kwacheon-shi, KOREA, REPUBLIC OF
Park, Mi Hyoun, Seoul, KOREA, REPUBLIC OF
Hwang, Sang Ho, Seoul, KOREA, REPUBLIC OF
Woo, Suk Gyu, Suwon-shi, KOREA, REPUBLIC OF
Song, Mi Kyung, Seoul, KOREA, REPUBLIC OF
Im, Jong Jun, Seoul, KOREA, REPUBLIC OF
Hong, Sung Gil, Seoul, KOREA, REPUBLIC OF
Kim, Joong Hark, Seoul, KOREA, REPUBLIC OF
Jang, Jung Soon, Seoul, KOREA, REPUBLIC OF
Kim, Hwa Young, Seoul, KOREA, REPUBLIC OF
PA EROMLIFE CO., LTD. (non-U.S. corporation)
PI US 2005130288 A1 20050616
AI US 2003-732340 A1 20031211 (10)
DT Utility
FS APPLICATION
LREP BURNS DOANE SWECKER & MATHIS L L P, POST OFFICE BOX 1404, ALEXANDRIA, VA, 22313-1404, US
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 937
AB The present invention relates to a novel Pediococcus genus microorganism and more particularly, Pediococcus pentosaceus EROM101 (KCCM-10517) originated from human intestines having immune enhancement, anticancer

and antiviral activities and a use thereof. Due to its excellent immune enhancement, anticancer and antimicrobial activities by activating macrophages/spleen cells and inducing gut immunity, the Pediococcus pentosaceus EROM101 of the present invention can be effectively used for the production of various products such as immune enhancement agent, anticancer agent, antimicrobial agent, food additive, intestinal function-controlling agent, live bacterial agent, feed additive and other fermented products.

L12 ANSWER 67 OF 84 USPATFULL on STN
AN 2005:138814 USPATFULL
TI Protein
IN Von Pawel-Rammingen, Ulrich, Lund, SWEDEN
Johansson, Bjorn, Lund, SWEDEN
Bjorck, Lars, Lund, SWEDEN
PI US 2005119464 A1 20050602
AI US 2003-499143 A1 20021217 (10)
WO 2002-EP14427 20021217
PRAI GB 2001-30228 20011218
DT Utility
FS APPLICATION
LREP NIXON & VANDERHYE, PC, 1100 N GLEBE ROAD, 8TH FLOOR, ARLINGTON, VA, 22201-4714, US
CLMN Number of Claims: 17
ECL Exemplary Claim: 1-26
DRWN 4 Drawing Page(s)
LN.CNT 1746
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A polypeptide isolated from S. pyogenes is described, having IgG cysteine protease activity. The protease is designated IdeS, Immunoglobulin G-degrading enzyme of S. pyogenes. A polypeptide comprises SEQ ID NO: 1 and variants and fragments thereof having IgG cysteine protease activity or the ability to generate an immune response against S. pyogenes in an individual. Polynucleotides encoding these polypeptides and the polypeptides may be used in generating an immune response in an individual. IdeS protease inhibitors may be used in the treatment of S. pyogenes infection.

L12 ANSWER 68 OF 84 USPATFULL on STN
AN 2005:124935 USPATFULL
TI Modified iRNA agents
IN Manoharan, Muthiah, Weston, MA, UNITED STATES
Kesavan, Venkitasamy, Woburn, MA, UNITED STATES
Rajeev, Kallanthottathil G., Cambridge, MA, UNITED STATES
PI US 2005107325 A1 20050519
AI US 2004-916185 A1 20040810 (10)
RLI Continuation-in-part of Ser. No. WO 2004-US11829, filed on 16 Apr 2004, PENDING
PRAI WO 2004-US7070 20040308
WO 2004-US10586 20040405
WO 2004-US11255 20040409
WO 2004-US11822 20040416
US 2003-493986P 20030808 (60)
US 2003-494597P 20030811 (60)
US 2003-506341P 20030926 (60)
US 2003-518453P 20031107 (60)
US 2003-463772P 20030417 (60)
US 2003-465802P 20030425 (60)
US 2003-469612P 20030509 (60)
US 2003-510246P 20031009 (60)
US 2003-510318P 20031010 (60)
US 2003-503414P 20030915 (60)
US 2003-465665P 20030425 (60)
DT Utility

FS APPLICATION
LREP FISH & RICHARDSON PC, 225 FRANKLIN ST, BOSTON, MA, 02110, US
CLMN Number of Claims: 89
ECL Exemplary Claim: 1
DRWN 28 Drawing Page(s)
LN.CNT 13589
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to iRNA agents, which preferably include a monomer in which the ribose moiety has been replaced by a moiety other than ribose. The inclusion of such a monomer can allow for modulation of a property of the iRNA agent into which it is incorporated, e.g., by using the non-ribose moiety as a point to which a ligand or other entity, e.g., a lipophilic moiety. e.g., cholesterol, is is directly, or indirectly, tethered. The invention also relates to methods of making and using such modified iRNA agents.

L12 ANSWER 69 OF 84 USPATFULL on STN
AN 2005:92902 USPATFULL
TI Human skin equivalents expressing exogenous polypeptides
IN Centanni, John M., Madison, WI, UNITED STATES
Allen-Hoffmann, Lynn, Madison, WI, UNITED STATES
PA Stratatech Corporation, Madison, WI, UNITED STATES (U.S. corporation)
PI US 2005079578 A1 20050414
AI US 2004-909119 A1 20040730 (10)
PRAI US 2003-491869P 20030801 (60)
US 2003-493664P 20030808 (60)
DT Utility
FS APPLICATION
LREP MEDLEN & CARROLL, LLP, 101 Howard Street, Suite 350, San Francisco, CA, 94105, US
CLMN Number of Claims: 93
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 4873
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to compositions for wound closure. More specifically, the present invention provides human skin equivalents engineered to express exogenous polypeptides (e.g., antimicrobial polypeptides and keratinocyte growth factor 2) and compositions and methods for making human skin equivalents engineered to express exogenous polypeptides. In addition, the present invention provides methods for treatment of wounds with human skin equivalents engineered to express exogenous polypeptides.

L12 ANSWER 70 OF 84 USPATFULL on STN
AN 2005:81051 USPATFULL
TI Microorganisms and cells for diagnosis and therapy of tumors
IN Szalay, Aladar A., Highland, CA, UNITED STATES
Yu, Yong A., San Diego, CA, UNITED STATES
Shabahang, Shahrokh, Redland, CA, UNITED STATES
Timiryasova, Tatyana, San Diego, CA, UNITED STATES
PI US 2005069491 A1 20050331
AI US 2004-485179 A1 20041105 (10)
WO 2002-IB4767 20020731
PRAI EP 2001-1184173 20010731
EP 2001-1259116 20011030
DT Utility
FS APPLICATION
LREP INTELLECTUAL PROPERTY / TECHNOLOGY LAW, PO BOX 14329, RESEARCH TRIANGLE PARK, NC, 27709
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 2864

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described are diagnostic and pharmaceutical compositions comprising a microorganism or cell containing a DNA sequence encoding a detectable protein or a protein capable of inducing a detectable signal, e.g. a luminescent or fluorescent protein, and, in a particular embodiment, furthermore (a) DNA sequence(s) encoding (a) protein(s) suitable for tumor therapy and/or elimination of metastatic tumors, e.g. a cytotoxic or cytostatic protein.

L12 ANSWER 71 OF 84 USPATFULL on STN
AN 2004:313935 USPATFULL
TI Wall teichoic acid as a target for anti-staphylococcal therapies and vaccines
IN Kokai-Kun, John Fitzgerald, Frederick, MD, UNITED STATES
Peschel, Andreas, Tubingen, GERMANY, FEDERAL REPUBLIC OF
Weidenmaier, Christopher, Tubingen, GERMANY, FEDERAL REPUBLIC OF
Kristian, Sascha A., Duesseldorf, GERMANY, FEDERAL REPUBLIC OF
PI US 2004247605 A1 20041209
AI US 2003-724194 A1 20031201 (10)
PRAI US 2002-430225P 20021202 (60)
DT Utility
FS APPLICATION
LREP FINNEGAN, HENDERSON, FARABOW,, GARRETT & DUNNER, L.L.P., 1300 I Street, N.W., Washington, DC, 20005-3315
CLMN Number of Claims: 44
ECL Exemplary Claim: 1
DRWN 10 Drawing Page(s)
LN.CNT 2361
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides vaccines comprising staphylococcal wall teichoic acid (WTA); vaccines comprising antibodies that specifically bind WTA; staphylococcal organisms deficient in WTA; and methods of treating patients suspected of having a staphylococcal infection.

L12 ANSWER 72 OF 84 USPATFULL on STN
AN 2004:286729 USPATFULL
TI Antimircobial peptide and methods of use thereof
IN Leung, Kai P., Libertyville, IL, UNITED STATES
Concannon, Sean P., St. Louis, MO, UNITED STATES
PI US 2004224897 A1 20041111
AI US 2004-795514 A1 20040309 (10)
PRAI US 2003-455206P 20030310 (60)
DT Utility
FS APPLICATION
LREP OFFICE OF THE STAFF JUDGE ADVOCATE, U.S. ARMY MEDICAL RESEARCH AND MATERIEL COMMAND, ATTN: MCMR-JA (MS. ELIZABETH ARWINE), 504 SCOTT STREET, FORT DETRICK, MD, 21702-5012
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 1417
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of preventing biofilm formation in an environment including the steps of administering to the environment an effective amount of a peptide having the amino acid sequence NH.sub.2-lys-lys-val-val-phe-lys-val-lys-phe-lys-CONH.sub.2. The method is useful in preventing the formation of biofilms in various environments including a home, workplace, laboratory, industrial environment, aquatic environment, animal body or human body. A method of inhibiting the growth of oral microorganisms including the steps of administering to an oral environment an effective amount of a peptide having the amino acid sequence NH.sub.2-lys-lys-val-val-phe-lys-val-lys-phe-lys-CONH.sub.2.

L12 ANSWER 73 OF 84 USPATFULL on STN

AN 2004:248186 USPATFULL
TI Use of xylitol to reduce ionic strength and activate endogenous antimicrobials for prevention and treatment of infections
IN Welsh, Michael J., Riverside, IA, UNITED STATES
Zabner, Joseph, Iowa City, IA, UNITED STATES
PA UNIVERSITY OF IOWA RESEARCH FOUNDATION, Iowa City, IA (U.S. corporation)
PI US 2004192786 A1 20040930
AI US 2004-766506 A1 20040128 (10)
RLI Continuation of Ser. No. US 2001-861841, filed on 21 May 2001, GRANTED, Pat. No. US 6716819
PRAI US 2000-205948P 20000519 (60)
DT Utility
FS APPLICATION
LREP MCKEE, VOORHEES & SEASE, P.L.C., 801 GRAND AVENUE, SUITE 3200, DES MOINES, IA, 50309-2721
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 6 Drawing Page(s)
LN.CNT 1172
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for killing infectious microbial cells by exposing the microbial cells to endogenous antimicrobial compounds. Activation of the antimicrobials is achieved by addition of low permeability, non-ionic osmolytes to lower ionic strength in body fluids where the antimicrobials have been previously suppressed by alteration of ionic transport (increase in salt concentration). The method can be used to treat cystic fibrosis. Cystic fibrosis causes elevated salt concentrations in the airway surface liquid (ASL) occur due to the impaired chloride transport across the epithelia. Xylitol has been found to be an effective low permeability, non-ionic osmolyte for use in the present invention.

L12 ANSWER 74 OF 84 USPATFULL on STN
AN 2004:232967 USPATFULL
TI Effectors of innate immunity determination
IN Hancock, Robert E. W., Vancouver, CANADA
Finlay, B. Brett, Richmond, CANADA
Scott, Monisha Gough, Vancouver, CANADA
Bowdish, Dawn, Vancouver, CANADA
Rosenberger, Carrie Melissa, Vancouver, CANADA
Powers, Jon-Paul Steven, Vancouver, CANADA
PI US 2004180038 A1 20040916
AI US 2003-661471 A1 20030912 (10)
RLI Continuation-in-part of Ser. No. US 2002-308905, filed on 2 Dec 2002, PENDING
PRAI US 2001-336632P 20011203 (60)
DT Utility
FS APPLICATION
LREP GRAY CARY WARE & FREIDENRICH LLP, 4365 EXECUTIVE DRIVE, SUITE 1100, SAN DIEGO, CA, 92121-2133
CLMN Number of Claims: 93
ECL Exemplary Claim: 1
DRWN 5 Drawing Page(s)
LN.CNT 6355
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of identifying a polynucleotide or pattern of polynucleotides regulated by one or more sepsis or inflammatory inducing agents and inhibited by a peptide is described. A method of identifying a pattern of polynucleotide expression for inhibition of an inflammatory or septic response. The method includes contacting cells with LPS, LTA, CpG DNA and/or intact microbe or microbial components in the presence or absence of a cationic peptide; detecting a pattern of polynucleotide expression for the cells in the presence and absence of the peptide, wherein the pattern in the presence of the peptide represents inhibition of an

inflammatory or septic response. Also included are compounds and agents identified by the methods of the invention. In another aspect, the invention provides methods and compounds for enhancing innate immunity in a subject.

L12 ANSWER 75 OF 84 USPATFULL on STN
AN 2004:220875 USPATFULL
TI Vaccine which comprises at least one antigen and a cathelididin derived antimicrobial peptide of a derivative thereof
IN Fritiz, Jorg, Vienna, AUSTRIA
Mattner, Frank, Vienna, AUSTRIA
Zauner, Wolfgang, Vienna, AUSTRIA
Buschle, Michael, Perchtoldsdorf, AUSTRIA
Egyed, Alena, Vienna, AUSTRIA
PI US 2004170642 A1 20040902
AI US 2003-344709 A1 20031014 (10)
WO 2001-EP9529 20010817
PRAI AT 2000-1416 20000817
DT Utility
FS APPLICATION
LREP Mark B Wilson, Fulbright & Jaworski, Suite 2400, 600 Congress Avenue, Austin, TX, 78701
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 10 Drawing Page(s)
LN.CNT 1624
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described is a vaccine which comprises at least one antigen and at least one cathelicidin derived antimicrobial peptide or a derivative thereof as well as the use of a cathelicidin derived antimicrobial peptide or a derivative thereof for the preparation of an adjuvant for enhancing the immune response to at least one antigen.

L12 ANSWER 76 OF 84 USPATFULL on STN
AN 2004:38136 USPATFULL
TI Antimicrobial agent
IN Bjorck, Lars, Lund, SWEDEN
Frick, Inga-Maria, Staffanstorp, SWEDEN
Schmidtchen, Artur, Lund, SWEDEN
PI US 2004028672 A1 20040212
AI US 2003-333319 A1 20030528 (10)
WO 2001-EP8208 20010717
PRAI EP 2000-306074 20000717
DT Utility
FS APPLICATION
LREP KALOW & SPRINGUT LLP, 488 MADISON AVENUE, 19TH FLOOR, NEW YORK, NY, 10022
CLMN Number of Claims: 41
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 1388
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of identifying an agent that enhances the anti-microbial activity of cationic anti-microbial peptides by blocking the inhibitory effects of the proteinase/glycosaminoglycan pathway, which method comprises: (i) providing, as a first component, a cationic anti-microbial peptide; (ii) providing, as a second component, bacteria; (iii) providing, as a third component, part of all of the components of a proteinase/glycosaminoglycan pathway such that the third component reduces the antimicrobial effect of the first component, for example, a glycosaminoglycan or bacteria or bacteria and a proteoglycan or a bacterial proteinase or a bacterial proteinase and a proteoglycan; (iv) contacting the first, second and third components with a test agent under conditions that would permit the killing of the bacteria by the

antimicrobial agent in the absence of the third component, and that would permit the inhibition of the anti-microbial activity of the first component by the third component in the absence of the test agent; (v) monitoring the survival of the bacterial culture thereby determining whether the test agent is capable of enhancing anti-microbial activity wherein a test agent capable of enhancing anti-microbial activity promotes killing of the bacterial culture. Agents identified by such a method are useful in the therapy of acute and chronic infections , particularly in the treatment of ulcers and in the promotion of wound healing.

L12 ANSWER 77 OF 84 USPATFULL on STN
AN 2004:24669 USPATFULL
TI Surrogate antibodies and methods of preparation and use thereof
IN Friedman, Stephen B., Chapel Hill, NC, UNITED STATES
PA Syntherica Corporation, Durham, NC, UNITED STATES (U.S. corporation)
PI US 2004018508 A1 20040129
AI US 2003-370052 A1 20030219 (10)
PRAI US 2002-358459P 20020219 (60)
DT Utility
FS APPLICATION
LREP ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
CLMN Number of Claims: 47
ECL Exemplary Claim: 1
DRWN 9 Drawing Page(s)
LN.CNT 4783
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A process is described for producing surrogate antibody molecules that mimic the structure, stability, and binding characteristics of a natural antibody. Surrogate antibody structure, composition of surrogate antibody libraries, methods of surrogate antibody preparation, and surrogate antibody applications are disclosed. Also disclosed are methods of surrogate antibody structural stabilization and resistance to nucleases. The surrogate antibodies comprise a specificity strand and a stabilization strand. The specificity strand comprises a nucleic acid sequence having a specificity region flanked by a first constant region and a second constant region. The stabilization strand comprises a first stabilization region that interacts with the first constant region and a second stabilization region that interacts with the second constant region. In further embodiments, the stabilization strand and the specificity strand comprise distinct molecules. In other embodiments, the surrogate antibody molecules may comprise polyoligonucleotides that have at least one nucleotide sequence that forms a loop with specific ligand-binding properties. Surrogate antibody libraries containing a large population of random binding molecules are pre-assembled and used in a process that captures and amplifies those molecules having prerequisite binding characteristics. The amplified surrogate antibody molecule produced by the process has identical structure and binding characteristics to the parent molecule captured from the initially assembled library. Surrogate antibody molecules contain binding loop(s) that are formed and stabilized by the hybridization of at least two adjacent and juxtaposed strands, one strand having a greater number of nucleotides than the other. The preparation of a polyclonal surrogate antibody reagent proceeds through phases of capture/enrichment and amplification, specificity enhancement, and affinity enhancement. Depending upon the intended application, polyclonal surrogate antibody reagents can be processed to monoclonality. These molecules expand upon the binding characteristics of natural immunoglobulins, and do not require animals, animal facilities, cell culture or the stimulation of an immune response, in their development. They can be used as an effective replacement for natural antibody molecules, and therefore can be used in testing methods like immunoassay, as therapeutic agents, for specific labeling, and for research purposes. Targets ligands compatible

with the development of surrogate antibodies include compounds, organisms, and cells that when complexed to a surrogate antibody in solution attain characteristics that can be physically or chemically differentiated from uncomplexed surrogate antibody.

L12 ANSWER 78 OF 84 USPATFULL on STN
AN 2004:12649 USPATFULL
TI Anti-pathogen treatments
IN Rider, Todd H., Littleton, MA, UNITED STATES
PA Massachusetts Institute of Technology, Cambridge, MA (U.S. corporation)
PI US 2004009167 A1 20040115
US 7125839 B2 20061024
AI US 2003-361208 A1 20030207 (10)
PRAI US 2002-355359P 20020207 (60)
US 2002-355022P 20020207 (60)
US 2002-432386P 20021210 (60)
DT Utility
FS APPLICATION
LREP HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 86 Drawing Page(s)
LN.CNT 9654
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Chimeric molecules that contain at least one pathogen-detection domain and at least one effector domain, and their methods of use in preventing or treating a pathogen infection in a cell or organism are described. The pathogen-detection domain and effector domain of the chimeric molecules are domains not typically found in nature to be associated together. Agents are also described herein having at least one pathogen-interacting molecular structure and at least one effector-mediating molecular structure, the agent being one that is non-naturally-occurring in a cell. The methods of prevention and treatment described herein are effective for a broad spectrum of pathogens and exhibit little or no toxic side-effects. Assays for the detection of a pathogen, pathogen component, or product produced or induced by a pathogen, are also provided.

L12 ANSWER 79 OF 84 USPATFULL on STN
AN 2004:1784 USPATFULL
TI Effectors of innate immunity determination
IN Hancock, Robert E.W., Vancouver, CANADA
Finlay, B. Brett, Richmond, CANADA
Gough Scott, Monisha, Vancouver, CANADA
Bowdish, Dawn, Vancouver, CANADA
Rosenberger, Carrie Melissa, Vancouver, CANADA
Steven Powers, Jon-Paul, Vancouver, CANADA
PI US 2004001803 A1 20040101
AI US 2002-308905 A1 20021202 (10)
PRAI US 2001-336632P 20011203 (60)
DT Utility
FS APPLICATION
LREP MORGAN & FINNEGAN, L.L.P., 345 PARK AVENUE, NEW YORK, NY, 10154
CLMN Number of Claims: 88
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 5838
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of identifying a polynucleotide or pattern of polynucleotides regulated by one or more sepsis or inflammatory inducing agents and inhibited by a peptide is described. A method of identifying a pattern of polynucleotide expression for inhibition of an inflammatory or septic response. The method includes contacting cells with LPS, LTA, CpG DNA

and/or intact microbe or microbial components in the presence or absence of a cationic peptide; detecting a pattern of polynucleotide expression for the cells in the presence and absence of the peptide, wherein the pattern in the presence of the peptide represents inhibition of an inflammatory or septic response. Also included are compounds and agents identified by the methods of the invention. In another aspect, the invention provides methods and compounds for enhancing innate immunity in a subject.

L12 ANSWER 80 OF 84 USPATFULL on STN
AN 2003:51669 USPATFULL
TI Virus derived antimicrobial peptides
IN Montelaro, Ronald C., Wexford, PA, UNITED STATES
Mietzner, Timothy A., Pittsburgh, PA, UNITED STATES
PI US 2003036627 A1 20030220
AI US 2001-785058 A1 20010216 (9)
DT Utility
FS APPLICATION
LREP BAKER & BOTTS, 30 ROCKEFELLER PLAZA, NEW YORK, NY, 10112
CLMN Number of Claims: 50
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 1450
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to peptides having antimicrobial activity (antimicrobial peptides). The antimicrobial peptides of the present invention are analogs of the Lentivirus Lytic Peptide 1 (LLP1) amino acid sequence. The invention is further directed to peptides referred to as the Lytic Base Unit (LBU) peptides derived from the LLP1 analogs, also having antimicrobial activity. In addition, the present invention is also directed to methods of using the peptides in a variety of contexts, including the treatment or prevention of infectious diseases. The antimicrobial LLP1 analog peptides and the LBU peptides (collectively eLLPs) may be highly active under high salt conditions and in biologic fluids. In addition, the eLLPs are effective when presented either in soluble form, or when attached to a solid surface. Furthermore, the peptides of the present invention are selectively active against a wide variety of bacterial pathogens and exhibit minimal toxicity to eukaryotic cells in vitro and in vivo.

L12 ANSWER 81 OF 84 USPATFULL on STN
AN 2002:330415 USPATFULL
TI Virus derived antimicrobial peptides
IN Montelaro, Ronald C., Wexford, PA, UNITED STATES
Mietzner, Timothy A., Pittsburgh, PA, UNITED STATES
PA University of Pittsburgh (U.S. corporation)
PI US 2002188102 A1 20021212
US 6887847 B2 20050503
AI US 2002-79075 A1 20020219 (10)
RLI Continuation-in-part of Ser. No. US 2001-785058, filed on 16 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2001-785059, filed on 16 Feb 2001, PENDING
DT Utility
FS APPLICATION
LREP BAKER & BOTTS, 30 ROCKEFELLER PLAZA, NEW YORK, NY, 10112
CLMN Number of Claims: 65
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 1553
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to peptides having antimicrobial activity (antimicrobial peptides). The antimicrobial peptides of the present invention are analogs of the Lentivirus Lytic Peptide 1 (LLP1) amino acid sequence. The invention is further directed to peptides referred to

as the Lytic Base Unit (LBU) peptides derived from the LLP1 analogs, also having antimicrobial activity. In addition, the present invention is also directed to methods of using the peptides in a variety of contexts, including the treatment or prevention of infectious diseases. The antimicrobial LLP1 analog peptides and the LBU peptides (collectively eLLPs) may be highly active under high salt conditions and in biologic fluids. In addition, the eLLPs are effective when presented either in soluble form, or when attached to a solid surface. Furthermore, the peptides of the present invention are selectively active against a wide variety of bacterial pathogens and exhibit minimal toxicity to eukaryotic cells in vitro and in vivo.

L12 ANSWER 82 OF 84 USPATFULL on STN
AN 2002:301730 USPATFULL
TI Virus derived antimicrobial peptides
IN Montelaro, Ronald C., Wexford, PA, UNITED STATES
Mietzner, Timothy A., Pittsburgh, PA, UNITED STATES
PI US 2002169279 A1 20021114
US 6835713 B2 20041228
AI US 2001-785059 A1 20010216 (9)
DT Utility
FS APPLICATION
LREP BAKER & BOTTS, 30 ROCKEFELLER PLAZA, NEW YORK, NY, 10112
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 1345
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to peptides having antimicrobial activity (antimicrobial peptides). The antimicrobial peptides of the present invention are analogs of the Lentivirus Lytic Peptide 1 (LLP1) amino acid sequence. The invention is further directed to peptides referred to as the Lytic Base Unit (LBU) peptides derived from the LLP1 analogs, also having antimicrobial activity. In addition, the present invention is also directed to methods of using the peptides in a variety of contexts, including the treatment or prevention of infectious diseases. The antimicrobial LLP1 analog peptides and the LBU peptides (collectively eLLPs) may be highly active under high salt conditions and in biologic fluids. In addition, the eLLPs are effective when presented either in soluble form, or when attached to a solid surface. Furthermore, the peptides of the present invention are selectively active against a wide variety of bacterial pathogens and exhibit minimal toxicity to eukaryotic cells in vitro and in vivo.

L12 ANSWER 83 OF 84 USPATFULL on STN
AN 2002:157591 USPATFULL
TI Novispirins: antimicrobial peptides
IN Lehrer, Robert I., Santa Monica, CA, UNITED STATES
Waring, Alan J., Irvine, CA, UNITED STATES
Tack, Brian F., Iowa City, IA, UNITED STATES
PI US 2002082195 A1 20020627
US 6492328 B2 20021210
AI US 2001-840009 A1 20010419 (9)
RLI Continuation-in-part of Ser. No. US 2000-606858, filed on 28 Jun 2000, PENDING
DT Utility
FS APPLICATION
LREP Pamela J. Sherwood, BOZICEVIC, FIELD & FRANCIS LLP, Suite 200, 200 Middlefield Road, Menlo Park, CA, 94025
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 1240
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novispirin peptides are antimicrobial agents with potent activity against Gram-negative bacteria. The peptides are nonhemolytic, exhibit reduced in vitro cytotoxicity relative to other antimicrobial peptides, and were well-tolerated in vivo after intravenous injection. Novispirins also bind lipopolysaccharide (LPS), a property that may mitigate symptoms associated with Gram-negative bacterial infection. A pharmaceutical composition comprising novispirin as an active agent is administered to a patient suffering from or predisposed to a microbial infection, particularly Gram-negative bacterial infections.

L12 ANSWER 84 OF 84 USPATFULL on STN
AN 2002:37948 USPATFULL
TI Use of xylitol to reduce ionic strength and activate endogenous antimicrobials for prevention and treatment of infections
IN Welsh, Michael J., Riverside, IA, UNITED STATES
Zabner, Joseph, Iowa City, IA, UNITED STATES
PI US 2002022668 A1 20020221
US 6716819 B2 20040406
AI US 2001-861841 A1 20010521 (9)
PRAI US 2000-205948P 20000519 (60)
DT Utility
FS APPLICATION
LREP ZARLEY MCKEE THOMTE VOORHEES & SEASE PLC, SUITE 3200, 801 GRAND AVENUE, DES MOINES, IA, 50309-2721
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 6 Drawing Page(s)
LN.CNT 1172
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for killing infectious microbial cells by exposing the microbial cells to endogenous antimicrobial compounds. Activation of the antimicrobials is achieved by addition of low permeability, non-ionic osmolytes to lower ionic strength in body fluids where the antimicrobials have been previously suppressed by alteration of ionic transport (increase in salt concentration). The method can be used to treat cystic fibrosis. Cystic fibrosis causes elevated salt concentrations in the airway surface liquid (ASL) occur due to the impaired chloride transport across the epithelia. Xylitol has been found to be an effective low permeability, non-ionic osmolyte for use in the present invention.